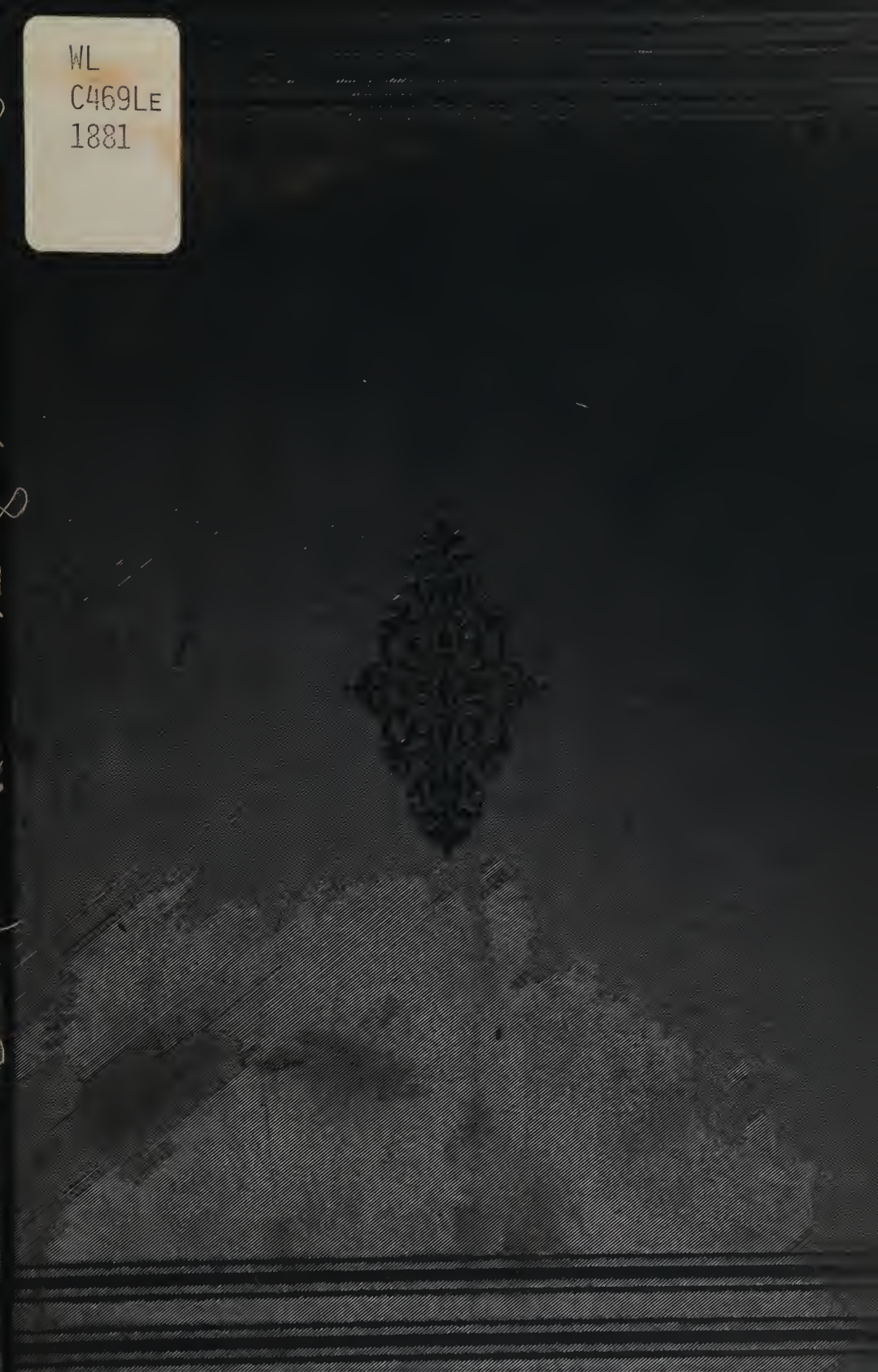

LECTURES

ON THE

PATHOLOGICAL ANATOMY

OF THE

NERVOUS SYSTEM.

DISEASES OF THE SPINAL CORD.

BY

J. M. CHARCOT,

Professor to the Faculty of Medicine of Paris; Physician to La Salpêtrière; Member of the Academy of Medicine; President of the Anatomical Society, and Ex-Vice-President of the Biological Society of Paris; Member of the Clinical Society of London, and Member of the Legion of Honor of France.

TRANSLATED FROM THE REPORTS BY DR. E. BRISSAUD,

IN THE PROGRÉS MEDICALE

BY

CORNELIUS G. COMEGYS, M. D.

Lecturer on Clinical Medicine to the Cincinnati Hospital; Honorary Member of the College of Physicians of Philadelphia; Member of the Cincinnati Medical Society; Honorary Member of the Historical Society of the Western Reserve of Cleveland, Ohio.

WITH ILLUSTRATIONS.

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PREFACE.

JUST thirty years ago Professor J. M. Charcot was kind enough to receive me as a private pupil in his service in La Charité Hospital, where he was a *chef de clinique*, and therefore able to offer very large facilities to foreign students who went to Paris for clinical study.

He held a high position at that early period of his career, as a clinician, and his instruction was very much sought for. His deliberate and thorough manner of procedure at the bedside aroused the attention of his pupils, who soon were made feel that the methodic investigation of disease requires the highest intellectual effort. It was not difficult at that time, even, to foresee for the young teacher a distinguished future. What he has done to develop the advance in the medical sciences during the last twenty years, I shall not attempt to detail. That he has enriched and enlarged the pathological and clinical field equal to any man of the age is generally conceded. More especially has he done his full share of the work that has brought again French medicine to the front line of modern progress. I believe that his genius, culture and special researches entitle him to be ranked with the celebrated men of modern times.

The translation of his lectures which I now present formed his course for 1879–80, and were reported in the *Progrès Medicale* by Doctor E. Brissaud. They have been published in the *Lancet and Clinic* of this city, beginning in September last. It has been desired that they should be republished in the present form.

I am sure that others might have made the translation better than myself, but no one could have done so with more reverence and grateful recollection for the eminent author.

C. G. COMEGYS.

Cincinnati, July, 1881.

AUTHORS REFERRED TO.

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LECTURES ON

THE

PATHOLOGICAL ANATOMY OF THE NERVOUS SYSTEM.

LECTURE ONE.

CONSTRUCTION AND SYSTEMATIC LESIONS OF THE SPINAL CORD.

Summary:—Retrospect and Present Course.—The Pathological Anatomy of the Nervous System is quite Incomplete.—A Decided Advance was made by the Discovery of the Lesion in Locomotor Ataxia; also in our Knowledge of the Disease known as Multilocular Induration.—Sclerose en plaques.—The Practical Character of Pathological Anatomy is shown in the better knowledge we have of the Pathology of the Nervous Centres in the Cerebro-Spinal Axis, and has greatly Amplified our Knowledge of the Construction of the Spinal Cord—A new Description of the Different Fasciculi of the Cord.—Lesions in the Gray Substance.—The Study of the Anatomy of Development, of Pathological Anatomy and of Symptomalogy, has led to the Discovery of the Existence of the New Anatomical Regions.—Revelations of the Cord at Birth.—The "Systematic" Tracts are Possessed of a Pathological Autonomy.—The Altered Functions of the Cord as Shown by Disease, could not be Demonstrated by Experimental Researches.—Symptoms Differ Accordingly as Different Tracts are Diseased.—Localization is the Dominant Idea in these Pathological Researches.—What is Meant by Localization?

GENTLEMEN:

Conformably to the programme which I have marked out, I must treat in the course of this year on the pathological anatomy of the nervous system.

Already on several occasions in this course of instruction which I began to-day, seven years since, I have had occasion to under-

take some demonstrations on various points relative to this great chapter.

Thus seven years ago appositely to inflammation considered in general, I presented an anatomico pathological sketch of *spinal scleroses*. The following year, while treating on degeneration and atrophies, I undertook to show the eminent role, and at that time but little known, which certain parts of the nervous centres play in the development of various trophic troubles and particularly in *muscular atrophies*. Finally, four years since, I essayed, in a series of lectures, to make plain the anatomico clinical method which shall permit us to establish on a solid basis the doctrine of *cerebral localization* in man. But on the whole, gentlemen, these diverse subjects have not been dwelt upon except in an incidental way, as elements contributing to the solution of more general questions. Moreover, notwithstanding the interest attached to them, they are only episodes in the anatomico pathological history of the nervous system. It seems to me that it would be useful now to consider in themselves the facts which compose this history both individually and collectively.

For the rest, the times appear to be favorable for undertaking this study. Innumerable materials, relative to these questions, have been laboriously collected in the course of a few years past. They have remained, as yet, scattered in various publications. The feeling has become general that they should be coördinated, in order to form a part of classical instruction. Thus, there have appeared recently, in France and elsewhere, several large monographs, and even some dogmatic treatises, concerning, especially, the pathology of the nervous system.

In these works the new documents always maintain beside those of the older periods an honorable place, indeed, they often take the first rank.

It will suffice, gentlemen, in looking over these works to see plainly the indications of a decided progress, and to establish that a great part in the accomplishment of this progress appertains to anatomico pathological researches, rendered more penetrating by the employment of the more perfect methods of modern histology. We dwell upon this point more particularly, gentlemen, by reason

of the special character of our instruction. To-day I wish to pause for a short time for its consideration. It is not a matter, in the account which I proceed to bring before you, of regular history, but solely an introduction, a summary, where will be signalized, very consipcuously, some of the principal results obtained. Proceeding in this way we shall reach possibly, and define the dominant idea which appears to have inspired these labors.

1. In the first place it is important to acknowledge, because an illusion under such circumstances is the worst of things, that notwithstanding all these efforts, there yet exists a considerable number of pathological states evidently seated in the nervous system, which leave no material appreciable trace on the cadaver, or revealed there, at the most, by the minutest lesions, without determinate character, incapable in any case to explain the principal facts in the morbid drama.

Such are, for example, tetanus and hydrophobia. The antique group of neuroses, though they may have been studied with considerable success on several points, yet continue to be nearly inaccessible to the anatomico pathologist. True epilepsy, paralysis agitans, hysteria even the most inveterate, in fine, chorea, exhibit themselves still as so many sphynxes which defy the most penetrating anatomy. We are also forced to confess on the threshold, that in the domain of neuro-pathology, pathological anatomy has a direct application in only a certain number of morbid states.

2. But let us take things as they are in reality, and confine ourselves solely to pathological forms in which the constant existence of a material lesion has been thoroughly established, and the field thus limited will still be vast enough.

It is often said that the progress of pathological anatomy and that of pathology go hand in hand; which is true, doubtless, in general, but it is not especially so in what concerns the diseases of the nervous system. Some examples will suffice to show that the discovery of a constant lesion in diseases of this nature is a result of great importance.

The description which has been given by Duchenne of Boulogne, of the symptoms of the affection which he has called locomotor ataxia is incontestably one of the most striking examples,—it is a

veritable *chef d'œuvre;* nevertheless, how much hesitation existed in the minds of practitioners until the time when the lesion described before by Cruveilhier, was, by the researches of Bourdon and Luys, allied to the clinical type. But some authors still thought that the affection in its origin might be a neurosis. All illusion, however, was dispelled when it was ascertained that the spinal lesion is already perfectly constituted and easily recognized in the first phases of the disease, when even it is not clinically revealed, save by a few fugitive symptoms barely recognizable. The ophthalmoscopic examination, which, in this species, answers in some sort to a vivisection, in determining the existence of a gray induration of the optic nerve, often years before the development of the other tabetic symptoms, sustains absolutely the same position. The lesion is therefore always present to a certain extent. It is not absent in the obscure, abnormal forms so varied and so different from the normal type. Its constant presence allows us unhesitatingly, to connect these forms, of which the number seems to augment every day, with the regular type seen only in the classical description of Duchenne.

The development of our knowledge relative to the disease known as *multilocular induration* of the nervous centres, *sclerose en plaques*, supports considerations of the same character. The disease has only been well known in its classical type when it has been connected with the cerebro-spinal lesion. The regular type is rare; the abnormal forms, on the contrary, are frequent and numerous. They could not be connected with the type from which they are separated clinically, except that pathological anatomy has served as a guiding thread.

In the examples which I have just brought before you, the intervention of pathological anatomy, and this is what I wish to be clearly understood, offers in some degree a character purely practical. The object especially, as you have observed, is to furnish to nosology for the determination of morbid compounds the more accentuated traits, more fixed, more material, so to speak, than do the symptoms themselves; no speculative idea intervenes and it requires but little effort to seize upon the nature of the relations which unite the lesions to the external symptoms.

Without misapprehending, gentlemen, the importance of the results obtained in this way, it is certain that to-day the study of the lesions can, without losing anything of practical force, be adapted to another point of view and assume higher aims and in some respects more scientific. They can, in other words, with the concours of experimental data, furnish the basis of a rational interpretation—physiological in fact, of the morbid phenomena.

3. This is what I desire now to make manifest to you, by marked examples chosen from among the new results introduced into the pathology of the nervous centres,—the brain, medulla oblongata, spinal cord, and I shall commence by what concerns the latter on account of its comparatively less complex construction.

(*a*) The anatomy of the spinal cord in the normal state, slightly magnified, does not present as you know, but, relatively, a simple structure. The transverse section of the inferior cervical region exhibits an axis of gray substance and a medullary covering. In the gray substance you distinguish, first, the anterior horns with the so-called motor cells, and the origin of the anterior nerve roots; then the posterior horns, where terminate the posterior roots; finally, you notice that the cornua are connected to each other by a commissure. In regard to the medullary envelope, which is formed by the mass of nerve tubes which have nearly a longitudinal direction, you distinguish two regions; first, the antero-lateral fasciculi, limited by the anterior groove and the collateral posterior groove; second, the posterior fasciculi limited by the commissure and the posterior horns. The space limited by the intermedian posterior grooves is not made of much account.

(*b*) The experimental method has not sensibly modified these data of descriptive anatomy, in the analysis of the functions of the different white fasciculi, and the several parts of the gray substance. It has distinguished the properties of the antero-lateral fasiculi taken as a whole,[1] also those of the posterior, and those of the two great regions of the gray substance, and it has not gone much beyond that.

(*c*) The methodic study of pathological lesions, you will soon see,

1 See work of Woroshiloff, 1875.

has demonstrated that the construction of the spinal cord is really much more complicated.

A great fact dominates, gentlemen, the *anatomy of the spinal cord;* it is the existence, very widely spread in this organ, of so-called *systematic lesions.* We understand on this subject, by the expression borrowed from the teachings of Vulpian, the lesions which settle in and circumscribe certain well defined regions without encroaching upon neighboring ones. I place before you a topographical scheme which exhibits the various regions which may be occupied by systematic lesions so far as yet known.

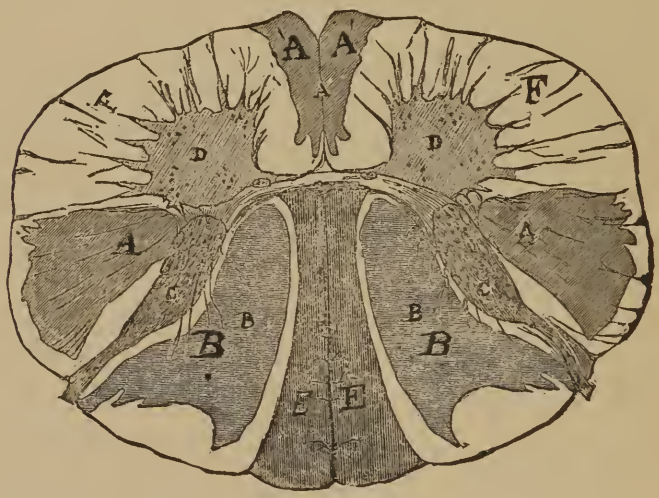

FIG. 1.—A, A, lateral columns. Á, Á, fasciculi of Turck. B, B, posterior radicular zone. C, C, posterior horns. D, D, anterior horns. F, F, anterior radicular zones. E, E, columns of Goll.

The posterior fasciculi considered in physiology as forming one whole, are, on the contrary, distinctly divided by pathological anatomy into two quite distinct parts. In this way the part adjoining the posterior groove, viz. : the tracts of Goll, may only be injured. In other cases the lesions relate to the posterior tracts adjoining the posterior cornua, that is to say, the radicular fasciculi (Pierret), the cuneiform fasciculi (Burdach).

The antero-lateral fasciculi may suffer from a degeneration of

like character. Thus, as a sequence of cervical lesions of a certain locality there is observed in the anterior part of the lateral fasciculi, in the neighborhood of the median fissure, a well defined lesion. It occupies a fasciculus of fibres (little or not all distinguishable in the normal state) and which extends from the medulla oblongata as far as the dorsal region of the cord. This is the fasciculus of Türck or the *direct pyramidal fasciculus*. This lesion of a direct pyramidal fasciculus is always accompanied by one of the same character, which occupies the posterior part of the lateral fasciculus of the opposite side in the same region always, and whose precise limits we shall have to study; this space responds to the *crossed pyramidal fasciculus*.

Between the base of the triangle, which represents a section of the crossed pyramidal fasciculus and the pia mater, exists on each side a space, under like circumstances untouched (at least in the cervical region); this space corresponds to the surface of a transverse section of the *direct cerebellar fasciculus* (Flechsig). These cerebellar fasciculi may themselves be systematically lesed.

Up to this time no one has seen examples of lesions relating to the region which surrounds the anterior cornua, and which alone continues in the normal state after this dissection effected by disease in the antero-lateral fasciculi. This region has received the name of the *anterior radicular zone* (Pierret), and the *fundamental region of the lateral fasciculi* (Flechsig).

Let me add, in regard to the gray substance, that there is quite a series of lesions, acute as well as chronic or subacute, which have the character of localizing themselves, systematically, in the anterior horns of the gray substance, where they affect, necessarily, the apparatus of the great motor nervous cells.

Thus, you see, from pathological anatomy, the old posterior columns decomposed into two secondary fasciculi, and the old lateral columns divided into three secondary fasciculi.

(*d*) But do the fasciculi that disease can thus isolate, as by a sort of selection, respond to so many regions of distinct anatomical systems, and at the same time endowed with particular functions? This is what seems to be now placed beyond doubt; on one hand by the anatomy of development of the spinal cord, and on the

other, by the study of the symptoms which reveal, clinically, these systematic lesions.

Let us consider the first point. It does not concern the first development, that which responds to embryonary phases, but the development of the cord at the epoch when the fœtus is at term or of the spinal cord of the new-born. After the very

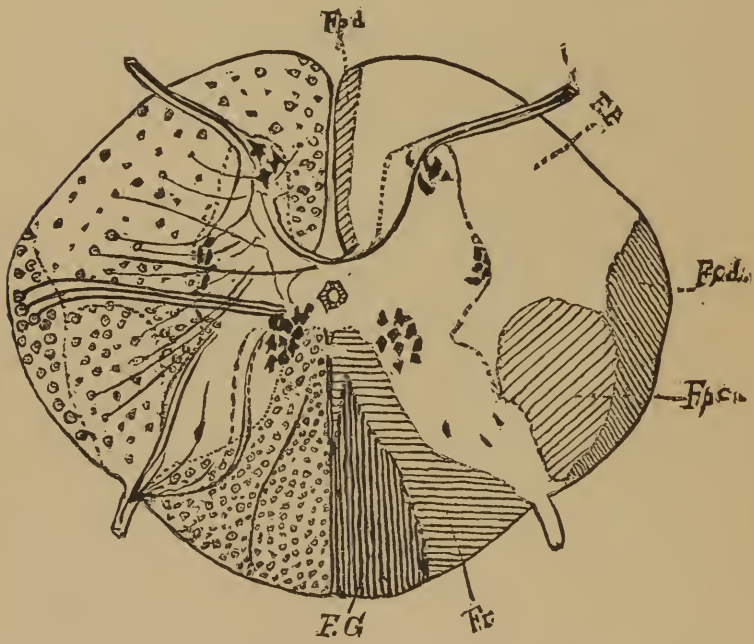

FIG. 2.—(From Flechsig). F.p.d., direct pyramidal fasciculus. P.F., fundamental part. F.c.d., direct cerebellar fasciculus. F.p.c., crossed pyramidal fasciculus. F.r., radicular fasciculus. F, G, fasciculus of Goll.

important studies of Flechsig and Pierret, which I limit myself simply to indicate here, because they will be the object of a regular description, all parts of the cord are not definitely constituted when the infant is brought forth.

Thus on the plate of Flechsig, which I now show you, and it relates to the cord of a new-born child, you can observe the following peculiarities, viz.: all parts shaded black are the ones developed; the axis cylinder is enclosed with its myeline sheath.

The undeveloped parts, by reason of the method of preparation,[1] remain, on the contrary entirely clear, because though the axis cyinder exists, the sheath of myeline is yet in default.

Now which are the parts that remain clear? On account of a coincidence which cannot be fortuitous, they are truly in the antero-lateral columns, the same crossed and direct pyramidal fasciculi that disease sometimes attacks separately.

According to the researches of Flechsig, as we shall see after awhile, these fasciculi would be in direct relation with motor regions of the cortex cerebri. Now these regions themselves, among animals which, the same as man, possess at the epoch of birth only automatic life, are not yet developed. It follows accordingly, that the pyramidal fasciculi can be considered as a sort of commissure reuniting the parts of the brain governing voluntary motion, with the parts of the cord governing automatic life.

Here is the topographic plan (see fig. 2) which brings together sufficiently exact, the studies of Flechsig on this interesting subject of the successive development of the spinal fasciculi. You will be able to assure yourselves, that it coincides in all points with the plan which has served to indicate the seat of spinal lesions (see fig. 1). In this way it will be easy for you to see, that the spinal fasciculi, in which are located systematic lesions, and possessed consequently of a *pathological autonomy*, are the very same of which the autonomy is signalized, also, by the study of development.

B. It is therefore more than probable, after the preceding considerations, that these same fasciculi are endowed with a functional autonomy. To make this clear we now refer to clinical observations. The observation has been recorded during the life of a patient, and after death, the anatomical investigation has been proceeded with conformably to the prescribed methods. Please remark that in this case it concerns systematic lesions distinctly limited, and that we assume, ideally, the place of the practical experimenter, when he endeavors to reproduce the lesion of the parts, which he supposes to have the special functions of which he undertakes the analysis. We can assert that, in the case of the spinal cord

1. Intervention of osmic acid.

the experimenter will be confounded with difficulties nearly insurmountable, which the systematic lesions resolve quite naturally. It would be impossible for even the most skilful experimenter to remove a spinal fasciculus in its whole length, an operation, moreover, which no animal could survive. Neither is it possible for him to attain, in a distinct manner, in the depths of the cord, the anterior cornua in order to destroy there, the mass of microscopic cells which they contain. Disease sometimes determines all these alterations, and most distinctly circumscribed.

I will add that the systematic spinal lesions are evolved most frequently by a chronic process, and their symptomatology is not complicated usually, by the reactionary phenomena of adjoining parts—phenomena which follow almost certainly experimental traumatism, and which render so difficult under such circumstances, the physiological analysis of the morbid phenomena.

But these phenomena which one attempts to eliminate by analysis,—and that, gentlemen, brings us back to plain pathology—constitute, justly, the symptomatology proper to each one of the lesions before us. I shall have to make you understand by what follows, that this symptomatology differs in reality, profoundly, accordingly as the lesion affects the pyramidal fasciculus, the cuneiform fasciculus, the anterior horns of the gray substance, and also according to the fashion of the lesion which occupies the fasciculi.

For the moment, I limit myself to show this fact, which I esteem fundamental in spinal pathology, that is, that the systematic diseases which we have just cited, should be considered as so many *elementary affections*, the thorough knowledge of which may be applied to the elucidation of more complex affections, non-systematic, or, in other terms, anatomically distributed in the nervous columns in a diffused and unequal manner.

Analysis directed conformably to these principles has not yet furnished all that it promises; nevertheless, I feel justified in declaring that it has contributed in a profitable way to the recent progress accomplished in the pathology of the spinal cord.'

I perceive that I shall be obliged to defer to an epoch, where I shall treat regularly these subjects, some considerations that I

desired to submit to you to-day, which concern the bulbar localizations, and localizations in the hemispheres of the brain.

If I have succeeded in placing in their true light the researches relative to the morbid anatomy of the nervous centres, you have not failed to recognize the leading tendency which is shown in all these labors. All seem, in some sort, controlled by what one might call the spirit of localization, which is, in fact, only an analysis.

The idea of localization is certainly not a new thing in pathological anatomy, it is as old as the science itself, though Bichat was the first, certainly, to formulate it definitely, while at the same time he developed from it all its scientific value; but, possibly, it had never been pursued with so much rigor and logic.

What, in short, do we understand by the term, to localize? In pathological anatomy to localize is to determine in the organs and in the tissues, the seat, the extent, the configuration, the palpable and material alterations. In physiological pathology it is interpreting the data in clinical observation and putting to profit the results of experimental research; to establish, in short, the relation between the functional troubles noted during life and the lesions revealed by the autopsy.

These two points of view will present themselves quite often in the course of our studies and they should be for us objects of equal attention; for, I repeat, gentlemen, it is not contemplative pathological anatomy alone, studying the lesion in itself and for itself, that we should know; it is, at the same time, pathological anatomy made available for nosology and for the clinic; in a word, applied to all the pathological problems within its range.

LECTURE TWO.

ON THE PYRAMIDAL FASCICULI.

Summary :—Principal Fact of Last Lecture is the Demonstration of Systematic Lesions of the Cord.—The Anatomy of Development at Birth a Field of Research.—Brain at Birth Less Developed than Bulb or Spinal Cord.—Outline of the Physical, Anatomical and Chemical Construction of the Brain in the New-Born.—Physiology of the Brain Corresponds to a Rudimentary State.—No Voluntary Power; all Action is Automatic.—Electrical Examination of Brain.—Difference in Development in Animals born Blind and those with Perfect Eyes.—The Anatomical and Physiological Facts have their Counterpart in Pathological States.—Absence of Symptoms.—Methods Employed to Bring into View Developed and Undeveloped Parts of Nerves.—Course of the Pyramidal Fasciculi.—Position of the Direct Cerebellar Fasciculi.—Columns of Turck.—Termination of Pyramidal Fasciculi in Great Cells of Anterior Cornua.—Pyramidal Fasciculi in Medulla.—Decussation Described.—Varieties.—Three Types of Asymmetries.—Relation of Hemiplegic Paralysis to (Asymmetrical) Decussation.

Gentlemen :

I dare hope that a principal fact is developed in the account which I presented to you at our last session, that is, that there exists in the domain of spinal pathology a certain number of diseases offering this remarkable character, that the lesion to which they are attached is fixed and cantoned, so to speak, in certain well defined regions of the nervous column; that these diseases constitute in some sort, so many elementary affections of which the profound study, should furnish precious documents for the elucidation of affections more complex, but anatomically not systematized.

These elementary or systematic affections, call them what you will, are those which logically, we must consider at once.

But before doing so, gentlemen, I wish to attempt to show you that certain documents drawn from normal anatomy, allow us to recognize as many distinct parts, anatomically and physiolog-logically, the same regions that pathological anatomy and the clinic have already placed in relief.

That demonstration I have presented to you as a sketch. To-day I wish to resume it, and press it so far, that you may be able to draw from it, in the point of view which we now specially consider, the numerous teachings that it includes.

These instructive facts, you know, we should seek, not in the anatomy of the adult, which in this regard furnishes data entirely insufficient, but rather in the anatomy of development. I will also remind you that it is not necessary to the end we have in view, to go back to the primary—the embryonic development, but that it suffices to consider the anatomical state of the diverse parts of the nerve-axis, as it is presented in the infant at birth.

I. A. It has been remarked for a long time, gentlemen, in the new-born, that while the spinal cord and the rachidian bulb are already relatively, in a very advanced development, it is far from being the case with the brain itself. The brain in the new-born, says Bichat, resembles but little that of the adult except in its exterior configuration, and it is now well-known that the principal details of its structure are barely outlined.

At this time of life, according to the description of Professor Parrot, which has become classic, the brain is a soft organ, gray colored, and uniform, in which the two substances, the gray and the white, are confounded. When we take in our hands a fragment of this nervous pulp and agitate it, it feels like a gelatinous mass of paste.

The histologic examination and chemical analysis have led to results which account in a great part for the macroscopic appearances. Everywhere the connective tissue, otherwise called the neuroglia, predominates; the reticulum is homogeneous and less distinctly fibrillar than in an adult. The cellular elements are present in large numbers, and their protoplasmic mass (as Parrot and Jastrowitz have shown) enclose physiologically, a certain quantity of fat in the form of granulations. On the other hand the nervous tubes are absent nearly everywhere, or, at least, barely outlined; here and there they are seen disseminated in the form of axis cylinders not yet covered with their coat of myeline. The chemical constitution responds to this anatomical state. The analysis of Schlossberger and those of Weisbach, have shown, for

example, that in the centrum ovale the proportion of water is represented by 92.59 per cent. in the cerebellum, and the pons by 85.77, and in the medulla oblongata by 84.38. Now the proportion of water is found precisely in the inverse ratio of the histological development.

To resume, in the infant at birth the structure of the brain is yet in a rudimentary state, while in the bulb and spinal cord it is endowed already with a structure that approaches the adult condition.

B. A glance at the physiology of the new-born, leads to considerations of the same character. In the infant at birth, says Virchow, reproducing a view already emitted by Billard, the life of the nervous system is in some respect exclusively concentrated in the bulb and in the spinal marrow. It is next to certain that voluntary determinations are then absent, and that accomplished acts, complicated as they may appear, suction for example, are purely instinctive or reflex in character.

The recent experiments of Soltmann, confirmed by Rouget and Tarchanoff, support the same view. In adult animals, contrary to the teachings of physiology ten years ago, the electrical excitation of so-called psychomotor regions determines movements in the members, or other parts of the opposite side of the body, and the ablation of the same regions, produces a state of paresis more or less pronounced, in the same members which just before moved under the electrical excitation. Now, it is shown by the researches of Soltmann, that the excitable parts of the cortex of the brain do not exist in the young of animals born blind, such as the rabbit and the dog, which like those of man are deprived of voluntary determinations; while by contrast, the excitable regions or motor centres, exist already, after the observations of Tarchanoff, in animals born with sight and endowed with voluntary movements. At the same time, according to these same researches, the brain of these animals presents a histological development and chemical constitution which differs but little from the condition of the adult state.

It may not be without interest to remark in passing, that these facts of anatomical and physiological order have their counter-

part in pathological states of the new-born. It is, for example, a matter well known by all authors who have particularly studied the pathology of that period of life, that all cerebral lesions, *even the most grave*, do not reveal themselves by any special symptom; properly speaking, they are latent and cannot be diagnosticated. Thus, Prof. Parrot expresses himself on the topic of cerebral lesions, which he subordinates to athrepsia, steatosis in focus, or diffused, white or red softening of the brain, intra-encephalic, or meningeal hæmorrhages, etc.

You see, gentlemen, at this age the brain does not yet exist in a

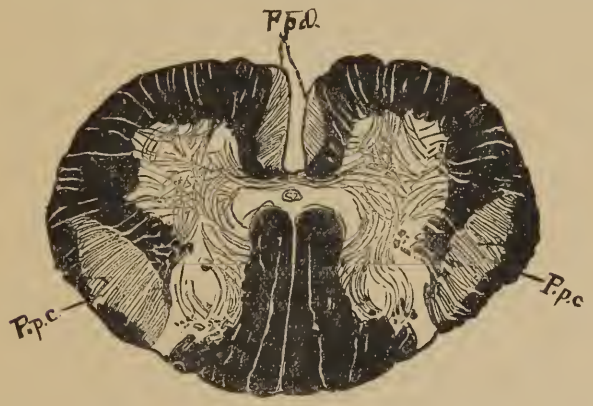

FIG. 3.—Cervical region. F.p.d., direct pyramidal fasciculus. F.p.c., crossed pyramidal fasciculus.

triple point of view—anatomical, functional and pathological; it is an organ indifferent.

II. The preceding considerations have served more or less to exhibit a contrast. In fact, it is well understood that the spinal cord, in an infant at birth, is much more advanced in development than is the brain itself; so much so, that in certain aspects it approaches the adult state. The same can be said of the rachidian bulb. However, and this is a point that concerns us now to bring out, the organization of these two parts of the spinal axis is still very imperfect. To convince yourselves of this examine the figures that I have already presented to you, and recall the methods employed to bring at once into view the differences which exist

between the developed and undeveloped parts. (See figures 1 and 2.) Osmic acid, I have told you, is fixed in the nervous elements covered by myeline. Now, you see on this section of the spinal cord, that four fasciculi very neatly defined, are unaffected by this reagent, which is equivalent to saying that these fasciculi are still undeveloped. They are: 1. The two fasciculi called *pyramidal direct;* 2. The two fasciculi called *pyramidal crossed.* And precisely these fasciculi correspond to the regions occupied by the lesion in one of the most interesting forms of systematic affections, which we propose to study in detail. This sufficiently indicates to you what interest we have in the anatomical study of

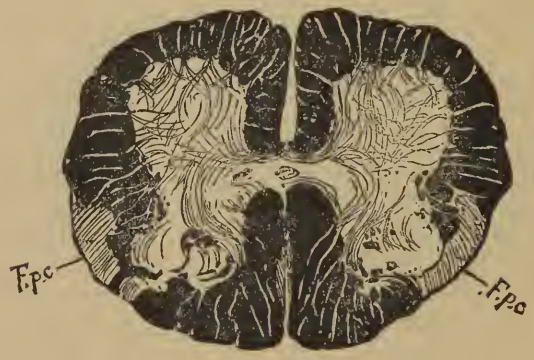

FIG. 4.—Lumbar region. F.p.c., crossed pyramidal fasciculus.

these fasciculi. They do not appertain exclusively to the spinal cord, we can follow them anatomically, and recognize their principal dispositions in the rachidian bulb, the cerebral peduncles, and even to the depths of the hemispheres. In this description we shall be guided by the labors commenced in Paris by Pierret, and, above all, by those of Flechsig. Lastly, we shall profitably make use of the studies of Parrot, founded on a hundred observations, the results of which will be presented to-morrow to the Society of Biology.

1. Let us see then, gentlemen, what course these four pyramidal fasciculi pursue, and we will commence, if it be agreeable to you, with the crossed fasciculi. (See figures 3 and 4).

Immediately below the bulbar decussation they occupy a position from which they do not change afterwards, and they can be traced to the inferior part of the cord as low down as the second or third sacral pair. In this great route they occupy the posterior half of the lateral columns, where they are represented by a compact fasciculus, triangular in form, touching in the rear the gelatinous substance, while within, a small space separates the summit of the triangle from the reticular process. The base of the triangle is directed outwards and is separated from the pia mater by a zone of nervous substance, forming a covering, which is composed of the *fasciculi cerebelli direct.* But this arrangement occupies only the superior moiety of the medullary column; below the dorsal region the cerebellar fasciculi disappear, and in the lumbar region, where there exists on sections trace of them, the pyramidal fasciculi touch the pia mater. On transverse sections the diameter of these triangular fasciculi diminishes regularly from above downwards and the fibres which compose them gradually disappear as they continue their downward course; but it is particularly at a level with the cervical and lumbar enlargements that this diminution in diameter is the most appreciable.

2. The anterior or direct pyramidal fasciculi, called also the columns of Türck, are located on the internal face of the anterior columns and present an ellipsoid form with the long axis antero-posterior. In general they can be traced as far as the middle of the dorsal region; but this disposition is subject to numerous exceptions; often they descend no further than the cervical region; sometimes, on the contrary, they descend as low as the lumbar.

These four fasciculi are composed of parallel fibres and when they are completely developed contain nervous tubes of all dimensions. How do they terminate? Since their diameter diminishes in proportion to their descent along the spinal cord, it is right to conclude that the nervous fibres which form them are averted successively on their pathway. The anterior horns of the gray substance appear naturally designated as being the points towards which these fibres converge. But do they penetrate the anterior roots? No, for the anterior roots and the nerve-cells of

the cornua are already greatly developed when the pyramidal fasciculi are immature. They do not pass either, at least, for the most part, into the commissures, which have also attained their development; then, they must terminate in the gray anterior substance, where they enter probably into connection with the great motor cells.

111. Next, gentlemen, we must attempt to follow the pyramidal fasciculi through the medulla oblongata.

In the first place, it is very easy to see that the spinal pyramidal fasciculi are nothing more than an emanation or prolongation of the bulbar pyramidal fasciculi.

Let a transverse section of the bulb be made at the median part of the olivary bodies. The location and connections of the pyramids are at once recognized, and it is seen that certain parts of the bulb have already attained an advanced development, such as the hypoglossal nuclei with the intra bulbar fillets of the hypoglossal nerves, and the whole field of the antero-lateral fasciculi.

Each one of these pyramids gives origin to two of the spinal fasciculi, one direct, the other crossed. The direct fasciculus descends in the interior of the anterior corresponding column; the other or the crossed fasciculus decussates with the corresponding fasciculus which proceeds from the other pyramid and gains the posterior part of the antero-lateral column, where it occupies the local relations that we have indicated heretofore.

In this way takes place the semi-decussations designated generally as the decussation of the pyramids, and of which all the particulars are described at length in classical treatises.

But what is less known about the fact of the intercrossing of the pyramids is, that it is subject to numerous variations, judging so at least from the observations of Flechsig, which cover nearly sixty examples.

The varieties in question, after Flechsig, may be grouped in three types:

1st. Type. This is the most common (75 per cent.) It consists in a symmetrical semi-decussation—each pyramid furnishing a direct and a crossed fasciculus. In the great majority of cases the direct fasciculus is much less important than the crossed

fasciculus. It is represented by 3.9 per cent. of the fibres of the pyramid, whilst the crossed fibres include 97 or 91 per cent. of the whole. But there exists in this type a very interesting variety which Flechsig has mentioned, and which Pierret has equally observed. It suffices to reverse the proportion that I have just stated; the direct fasciculus is represented by 90 per cent. of all the fibres, while the crossed fasciculi contain only about 10 per cent. of the whole number. The number of the intercrossed fibres in such a case are scarcely worth being taken into account.

You comprehend, gentlemen, all the interest of a case of this

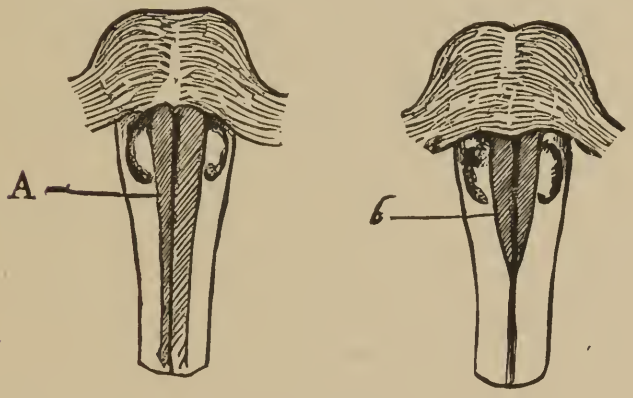

FIG. 5 and 6.—A predominance of the direct pyramidal fasciculus; b, type of total decussation. The direct pyramidal fasciculus is in default.

character in the point of view of direct cerebral paralysis. You readily understand how that such paralyses, contrary to the rule, are produced on the same side as the lesion.

The existence of these paralyses is incontestable, but certainly are seen less frequently than has been claimed nowadays. The number of two hundred that has been displayed in order to sustain the needs of certain theories, could be very considerably reduced by a critical examination. Nevertheless, some observations, carefully collected by Brown-Sequard, Callender, Jackson, Reynaud and others, leave no doubt of their existence.

Generally, to explain these facts, the theory of Longet is

invoked, who supposes under such circumstances, the intercrossing is in default; but this was, on Longet's part, only pure hypothesis. Indeed, the anatomists have always considered the intercrossing of the pyramids as a disposition absolutely constant. On this point Serres affirms to having examined 1100 subjects without finding a single exception to the rule. Well, gentlemen, that opinion appears greatly too absolute, and the researches of Flechsig, demonstrate it to be so. In many cases, if the intercrossing is not absolutely in default, it may, as I have said, be represented only by a few fibres—so restricted that they may be regarded as of little importance, and, in that case, the direct fasciculus takes the place, almost completely, of the crossed fasciculus; a sufficient circumstance to explain the direct paralyses.

2d type. This has been observed 11 times in 100. It is the total decussation; in other words, the direct fasciculi fail completely.

3d type. This is more frequent than the preceding, since it has been observed in the proportion of 40 per cent. ; it should merit the designation of asymmetric type. In such a case there exists but three fasciculi; one only of the pyramids is divided into two fasciculi, one direct, the other crossed; the second pyramid, on the contrary, is intercrossed *in toto.*

Finally, gentlemen, there remains for me to signalize the remarkable relation of compensation which exists between the two fasciculi issue of one sole pyramid, according to the cases to which I have just referred. The more the one is voluminous, the more the other is reduced and *vice versa.*

This asymmetry in the decussation continues its course in the cord and corresponds to asymmetries there. It is important to know this, because one might at first view in a certain case, consider it as a pathological condition.

LECTURE THREE.

ON THE PYRAMIDAL FASCICULUS IN THE CEREBRAL PEDUNCLES, INTERNAL CAPSULE AND CENTRUM OVALE.

Summary:—Section of Peduncles above the Pons.—Construction of the Peduncles.—Crusta, Tegmentum, Locus Niger and Red Nucleus of Stilling.—Division of Crusta into Three Segments.—The Pyramidal Segment at the Period of Birth.—Pyramidal Tract in the Internal Capsule; also in the Centrum Ovale.—Its Termination in the Rolandic Region of the Cortex.—Hypothesis of Flechsig in Regard to its Embryonic Development.—Chromotological Demonstrations by Parrot in the Anatomy of Development.—Comparison of the Views on that Subject of Flechsig and Parrot.

GENTLEMEN:

In the last lecture, basing my remarks principally on the researches of Flechsig, yet controlled by my own observations, we were able to recognize the topography of the pyramidal fasciculi in the various regions of the spinal cord, and to indicate the relations they sustain to other constituent portions of this acknowledged complex organ. Then rising above the decussation we have further found in the bulb these pyramidal columns reunited in two very distinct fasciculi already known to you in descriptive anatomy as the *anterior pyramids*. To-day, gentlemen, we must go still higher and determine as far as possible the course of the pyramidal fasciculi in the other parts of the isthmus, that is to say, in the protuberance, in the cerebral peduncles and, finally, in the brain itself, where, as you will see, they appear to have their origin.

1. In the protuberance or pons, the nervous elements which have formed the pyramids of the bulb, do not appear any longer as compact fasciculi; they are disassociated and intermingled with the proper protubernantial fibres, where they contribute to form a network, in the midst of which, it is, at least, very difficult to distinguish them.

It is not so, however, in the cerebral peduncles, where we shall see them reconstructed to a condition of very limited fasciculi.

2. You all know, gentlemen, that those two columns which connect the protuberance to the cerebral hemispheres are called the cerebral peduncles. I assume that a section has been made of them perpendicularly to the direction of the fibres, which are so plainly seen on the inferior face of these columns and a little above the protuberance, on a level with the origin of the third pair of nerves. Let us study first the peculiarties which a similar section would show in an infant at full term. The peduncle is divided by authors into two parts, viz.: 1, the inferior layer which is called

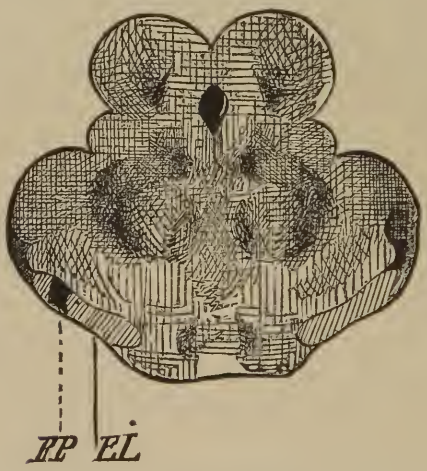

FIG. 7.—Schema of a section of the peduncles in an infant at birth (Flechsig). E.L., inferior layer, crusta. F.P., pyramidal fasciculus.

the *crusta;* 2, the superior layer, the *tegmentum*, cap, or haube, as denominated by the Germans. Above these parts in this section we observe the *tubercula quadrigemina anterior.* Finally, towards the median part, you see the lumen of the aqueduct of Sylvius, around which is developed a mass of gray substance representing the anterior cornua of the spinal cord, and there also are disposed cellular groups in which the *motores oculorum* take root.

In advance of this point we distinguish the prolongations of the

anterior spinal fasciculi (the fundamental part of the antero-lateral fasciculi); these are the fibres which are intermingled with those of the *processus cerebelli ad testes.*

These processes are seen on the plane of the section as red spots (rothe kerne of Stilling.) The diverse parts, that constitute, properly speaking, the *tegmentum*, are already developed in the

FIG. 8.—Horizontal section of the peduncular region in a case of secondary degeneration. T, superior layer (tegmentum). F, inferior layer of sound side. L, locus niger. P, internal fasciculus of the lower layer of the diseased side. D, secondary degenerations occupying nearly the median two-fourths of the base.

NOTE.—In this figure and especially in the preceding one, we observe that the region of the pyramidal fasciculus occupies but about a fourth part of the inferior layers (third quarter proceeding from within outwards). Such is at least the extent assigned to this fasciculus by Flechsig. I must say that a certain number of examinations of the peduncular region in the new-born, has led me to think that this region is more extended than Flechsig would give us to understand. So far as relates to secondary degenerations in the pyramidal fasciculi, I can, in a measure, affirm that they correspond to dimensions, sensibly, much greater than his researches would lead us to to suppose. A number of anatomical observations collected latterly in my service at Salpétriere do not lead me to have the least doubt about it. It results from these observations, that the pyramidal fasciculi occupy, at least, the two middle fourths of the crusta.

new-born, and you are aware that in ascending towards the encephalon, we shall see them terminate in the optic beds, where they disappear. Thus they form no part of the peduncular expansion. I have paused to describe all of the structures together, in order to better determine the topography of the region which I must next display to you. Henceforth we can consider them abstractly.

11. It is of more import, on the other hand, to study attentively the inferior layer (*crusta*) of the peduncles. In the adult it is distinctly separated from the superior layer (*tegmentum*) by a transverse band of gray substance, which is the *locus niger* of Sœmmering. The nervous cells of this region are not infused with pigmentary matter at the period of life which we are studying. Be that as it may, it is below this transverse band that we find the region of the crusta and it is there that we must search for the tracts of the pyramidal fasciculi.

A section of the inferior layer, crusta, of the peduncle, may be divided into three segments of nearly equal dimensions, viz.: the internal, external and median. In the first two, the nerve tubes are not yet invested with their medullary envelope and appear in the section as clear spaces. It is not the same in the median segment. This is seen as a space, rhomboid in form and opaque, because at this point the nerve fibres have acquired a complete development. Now it is precisely this opaque substance that, according to Flechsig, represents the reconstructed prolongations of pyramidal fasciculi. (See note in relation to Fig. 8). Thus you see that while in the cord and the bulb, of the new-born, the pyramidal fasciculi are distinguished from the neighboring parts by reason, even, of the rudimentary state to which their clear coloration is due; it is quite the contrary, in the cerebral peduncles. There, the nerve fibres, which compose the pyramidal fasciculi, are already invested with myeline and they show, consequently, the marks of an advanced development which puts them in contrast with surrounding parts.

Here then, gentlemen, we have an incontestable and remarkable fact, since it seems to indicate that the development of the pyramidal fasciculi proceeds from the brain itself. M. Flechsig

has not failed to give it due prominence. He has even been led by his observations to emit the hypothesis that it is in the gray substance of the cortex, or, in other words, in the ganglionic cells which are found there, that the nerve fibres of the future pyramids take their origin; there they begin to appear in the form of buds or shoots, which develope progressively, and descend gradually, in the peduncles, and, after having traversed the protuberance and the bulb, reach the spinal marrow, where they finally terminate in its inferior extremity. I leave, let it be understood, to M. Flechsig the responsibility of his hypothesis, and I proceed hereafter to limit myself to the exposition of the data on which it rests, not having had yet occasion to verify personally this latter part of his researches.

3. Following Flechsig, you have foreseen, that the pyramidal fasciculi may be followed beyond the crusta into the depths of the hemispheres. We can, in the first place, recognize their presence in the midst of the opto-striated ganglionic masses in the region that is called the *internal capsule*, and which, for a considerable part at least, is nothing else than the expansion of the fasciculi which form the lower layer (crusta) of the peduncles. In regard to the internal capsule a few topographical indications will not be superfluous.

If we make a horizontal section of one of the cerebral hemispheres, following a line parallel to the *lateral posterior fissure* of Henle, and a little above it, then the inferior segment of the hemisphere which is thus displayed, offers for our study the following particulars: behind and within, close to the median line, is the optic bed; in front of this is the head of the nucleus caudatus; without, the lenticular nucleus of which the internal borders form, by their reunion, a sort of wedge, which juts into the angle formed by the nucleus caudatus and the optic bed. But the internal ganglionic masses (thalamus opticus and corpus striatum) are separated from the external mass (*nucleus lenticularis*) by a large white, bent tract which is the internal capsule, or, in other words, to a certain extent, the peduncular expansion.

You will recognize, gentlemen, following the topographical position of this tract, that it is composed of two very distinct parts: 1,

the anterior part comprised between the internal and anterior face of the lenticular nucleus and the head of nucleus caudatus; 2, a posterior part, intermediate to the external face of the optic bed

FIG. 9.—Horizontal section of the right hemisphere parallel to the fissure of Sylvius. N.c., nucleus caudatus. S.a., anterior segment of internal capsule. C.o., optic bed. F., circumscribed focus in the posterior segment of the capsule and occupying a part of the intra hemispheric tract of the pyramidal fasciculus.

and the postero-internal border of the lenticular nucleus. These two parts are reunited in the form of an obtuse angle which may be called, after Flechsig, the knee of the internal capsule.

Now, it is in the posterior segment of the internal capsule, according to Flechsig, that we must seek the origin of the intra-hemispheric tract of the pyramidal fasciculus.

At the period of its growth, in which we study it, this fasciculus is seen in the posterior segment as an eliptical space, in sharp contrast by its opacity with the adjoining parts; therefore composed of nervous fibres already well developed. It has no relation of continuity with adjacent ganglionic masses; and if we make an ideal division of the posterior part of the internal capsule into three regions of equal extent, we shall find the middle one occupied by the pyramidal fasciculus. Moreover, beyond the internal capsule the pyramidal fasciculus may be followed as far as the middle parts of the *centrum ovale* and even unto the gray couches of the cortex cerebri.

4. To obtain a view of the pyramidal fibres in this last part of their tract, we must examine a frontal section of the brain. This section made slightly in the rear of the fissure of Rolando, and parallel to its direction, divides, in the middle, the ascending parietal convolution, which, in Pitres topographical system, is called the parietal section. It reveals the posterior segment of the internal capsule, directed obliquely upward and outward, the lenticular nucleus forming the external border, the optic couch cut transversely through its centre bordering its internal side; and above it is surmounted by the cue of the striated body. It is precisely here in this posterior segment where, according to Flechsig, the pyramidal fasciculi pass upward. Reaching the centrum ovale, they begin to separate and scatter so widely that it is impossible to follow them.

Nevertheless a portion of the fasciculus remains coherent and continues its path towards the superior extremity of the central convolutions. Thus, in leaving the capsular region, they diverge outwards in such a manner as to circumscribe the side of the ventricle, turning slightly upwards, they afterwards pursue their vertical course towards the termination which we have just pointed out.

In sum, gentlemen, that part of the gray cortex, in which these terminal fibres of the pyramidal fasciculus end, appertains, you now see, to the region that is designated as the motor zone (lobule

paracentral, superior extremity of the frontal and parietal ascending convolutions).

Such are the results of the researches of Flechsig in what concerns the intra-cephalic tract of the pyramidal fasciculus. On more than one point, these results seem to require further confirmation, and the author himself does not appear to accord to them his absolute confidence. But what is necessary to retain of them is

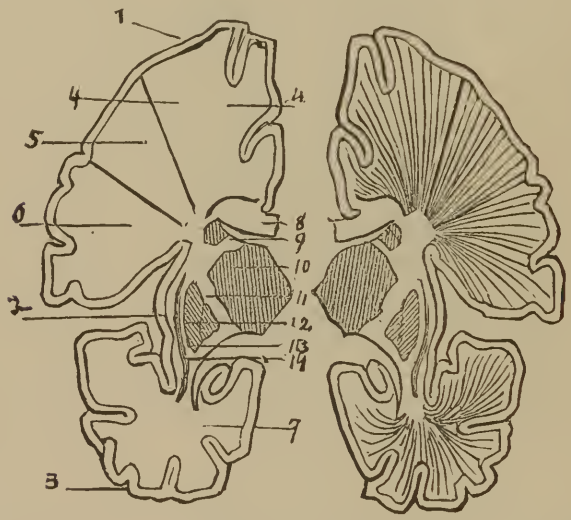

FIG. 10.—Parietal section of the hemisphere (Pitres). 8, corpus callosum. 9, end of the caudate nucleus. 10, optic couch. 11, internal capsule (posterior segment). 12, lenticular nucleus. 13, external capsule. 14, outer wall, claustrum.

the grand range of that tract which we have successfully studied in the diverse regions of the cerebro-spinal axis.

The hypothesis to which Flechsig appears to have been led, from the sum of the facts observed by him is, that the pyramidal fibres have their origin in the cortical substance of the motor zone; there they begin to develop, and thence descend by a shooting or budding process to form the pyramidal fasciculi, in the capsule, the peduncle, the pons, the medulla oblongata, successively, and according to the progressive age of the child, in the most inferior parts of the spinal axis.

5. But at this point, gentlemen, it is proper to introduce the researches of Professor Parrot, of which I made mention in my last lecture. These researches, communicated very recently to the Society of Biology, do not accord precisely, with the views presented by Flechsig, but they approximate them, and assist to make them more distinct. If, in a general way, they confirm the results announced by Flechsig, they tend at the same time to modify them on an important point—I refer to the intra-cephalic tract about which the German author formulates the hypothesis as shown above.

The researches of Parrot are founded on ninety-six autopsies of infants who were not over a year old. The brain was examined by methodic sections, on the surface of which it was the object to see, by the naked eye, the differences in color, which correspond to the diverse regions of the cerebral mass, according to the age of the infant. It is certain that these *chromological* facts, as they are called by Parrot, can furnish indications relative to the development of the different parts of the hemispheres. The gray parts, transparent, indeed, may be considered as fœtal or embry-

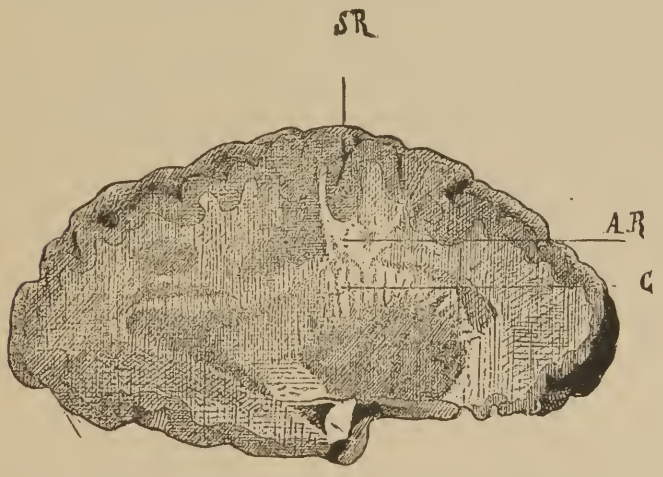

FIG. 11.—Vertical antero-posterior section slightly in advance of the internal face of the hemisphere. The parts are diversely shaded, some white, others gray. A. R. represents the Rolandic coil subjacent to the fissure of Rolando S. R. C, internal capsule emerging from the grey central mass.

onary, while the white portions are the adult or developed ones. The white coloration, in a word, corresponds to the completed structure of the nerve tubes (axis cylinders invested with their myeline.) At this point, it seems to me, interesting to refer more especially to the important researches of Parrot.

This figure, which Parrot has very obligingly placed at my disposal, represents a vertical antero-posterior section of a hemispere parallel to the median plane and a half an inch from the inter-hemispheric fissure. It relates to an infant of seventeen days. You observe that, at this epoch, the anterior and posterior regions have a very pronounced gray coloration, which indicates that they are yet in the fœtal state; it is only at the end of a month that the substance of the occipital lobe begins to whiten; and only after four months, and close to the fifth, do the anterior regions begin to be developed, and this development will not be accomplished until towards the ninth month.

But already, you observe, about the seventeenth day, the median part, or sub-Rolandic, of the brain, is marked by the presence of nervous fibres, clothed with myeline. At this time, the fasciculi of the centrum ovale are seen in the form of two white tracts which seem to proceed towards each other. Only the oldest, judging from the study of the brain of younger infants, rests upon the central ganglionic masses, which seem to be its centre of formation, and it advances in the direction of the furrow of Rolando; the other forms a species of white coil, circumscribing the depression of the furrow of Rolando below the gray cortex (Parrot calls it the *coil Rolandic*), and stands in advance of the preceding fasciculus.

These white tracts, you clearly see, respond entirely, in their anatomical relations, to the description given by Flechsig of the intra-hemispheric tract of the pyramidal fasciculi; only, and it is here that the opinion risked by Flechsig is impaired by the results obtained by Parrot, there should exist, for this part of the pyramidal fasciculus, two formative centres, the one seated in any point of the central nuclei and it would be first in point of age; the other would have its point of departure in the gray substance of the Rolandic convolutions, or, in other words, the motor zones. In

short, I repeat, there is but little difference, except on this point, between the two observers, and we may henceforth conclude from the researches of Flechsig that, of all the regions of the cortex of the hemispheres, it is the motor ones, so called, that are first developed and are the first to place themselves in relation with the bulbo-spinal system, by the intermediation of the pyramidal fasciculi.

Here terminates the considerations that I have thought proper to present to you, concerning the anatomy of the system of the pyramidal fasciculi, according to the documents furnished by the examination of the development of the nervous centres. This preliminary exposition, possibly too long, was indispensable, in order to undertake the study of systematic lesions of the pyramidal fasciculi, known as secondary degenerations. These lesions are not interesting in the sole point of view of pure pathological anatomy; they are related to a multitude of clinical facts which render them worthy the attention of medical men.

LECTURE FOUR.

SECONDARY DEGENERATIONS.

Summary:—Systematic Lesions of the Spinal Cord.—Secondary Degeneration in the Pyramidal Fasciculi are, usually, Consecutive to Focal Lesions in the Brain, or Spinal Cord; they are Called Descending Degenerations.—All Lesions in the Cerebral Cortex do not Produce Descending Degenerations.—Localization Dominates the whole Question.—The Cerebral Foci that Produce Descending Degeneration in the Pyramidal Fasciculi Must be Localized in the Rolandic Area.—The Lesion Must also be Destructive in Character.—Description of the Degeneration of the Pyramidal Fasciculi, as Seen in the Peduncles, Pons, Medulla Oblongata and Lateral Columns of the Cord.—Description of the Internal Capsule and Localization of the Pyramidal and Other Regions Therein.

GENTLEMEN:

After the preliminary studies with which we have been occupied, we are now prepared to enter upon the domain of pathological anatomy. I propose to develop before you the systematic lesions of the spinal cord. I will begin the study of the diverse forms which compose this class, with those alterations generally known as *secondary degenerations*.

I. The lesions in question are called secondary, because they are produced, most often, consecutively to other lesions of a focal character which are, primarily, seated in different regions of the cerebro spinal axis—the brain, the bulb, the spinal cord, or even in the peripheral nerves. Once begun these consecutive lesions can acquire an individuality—a real autonomy, and there is attached to them, at times, a clinical history which is superadded to that of the original disease, and occasionally even dominates it.

In this point of view the most interesting lesions are those which affect the system of the pyramidal fasciculi. I will state in advance, gentlemen, that all of the systematic lesions of the pyramidal fasciculi are not secondary; there are lesions of this kind (and on this point I shall dwell by and by) which are developed in

a primitive protopathic manner, that is, outside of all influence from a primordial lesion.

I cannot too strongly, insist, gentlemen, upon the point, that the history of secondary spinal degenerations is entirely worthy to fix the attention of physicians. If indeed these lesions have for a long time been considered as objects of pure scientific curiosity, only proper to interest the physiologist or anatomist, I can affirm that it is not so now. Recent and laborious investigations have sufficiently placed beyond doubt, that such affections hold, in every point of view, a most important place in the pathology of the nervous centres.[1]

II. The secondary lesions of the spinal cord are numerous, and complex enough to require a separation into divisions:

1. In the first group are found *secondary degenerations of cerebral origin*, that is, those that proceed from focal lesions occupying certain parts of the encephalon. Thus, the lesions of this group may result, (*a*) either from a primitive lesion of the brain itself, (*b*) or from lesions situated in various regions of the isthmus (the peduncles, protuberance, medulla oblongata). The degenerations which have thus originated, are commonly called descending, because they seem, really, to descend from the encephalon where they begin, towards the peripheral parts.

2d. In the second group we must place *secondary degenerations of spinal origin*, that is, those which are consecutive to the formation of a focal lesion in any point of the cord. We shall soon see why these consecutive lesions may be called at one time descending, and ascending at another.

3d. A third category includes a small number of facts very curious too, but which have not yet found a place in practice. The degeneration here has a peripheral cause and is located in the posterior fasciculi of the spinal cord. The examples known amount to five or six; but always they are nerves of the cauda equina, and, most explicitly, the posterior

1 It is scarcely necessary to refer to this again, we recall only that after the labors of Türck, Charcot and Vulpian, it is in the classical memoir of Professor Bouchard that we will find the most interesting documents relative to secondary degenerations. Bouchard was the first to make use of the processes of hardening and coloration in the investigations of medullary lesions, and the first, moreover, to investigate and make known the symptomatology of degenerative lesions.

roots are considered as the point of departure of the special lesion.

III. We will occupy ourselves, in the first place, with the degenerations, of the first group, with those which have a cephalic origin, and, even, to limit still more our first field of study, we will begin with secondary fascicular lesions, developed only from a focal point, seated in the brain itself.

Before entering into this detail let us indicate the more general characters of the group.

1. Let it be well understood, that it is not a matter of indifference in what part of the hemisphere it is seated. In other terms, there are extensive regions in the hemispheres where a focal lesion is incapable of producing secondary degenerations; and there are others, on the contrary, where these lesions determine with positive certainty, consecutive descending alterations. You see, gentlemen, that a question of localization dominates, indeed, the situation.

2. As to the *nature* of the original lesion nothing is more variable. Tumors intra, or extra cephalic, foci from hemorrhages or softening, in short, all possible alterations will have the same effect, provided always, and it is a condition *sine qua non*, that the lesion be *destructive*, that is to say, produces in the place where it is established a veritable loss of substance at the expense of the nervous elements. Thus, chronic foci, from hemorrhage, or softening, figure much more frequently among the lesions which cause secondary degenerations than tumors generally do, as these may compress or displace nervous elements without destroying them.

3. Beyond a particular case which will be mentioned at the proper time and place, degenerations of this group, as a general rule, affect exclusively the system of the pyramidal fasciculi and do not injure adjoining tracts.

If the primordial lesion occupies but one of the two hemispheres, the degeneration is propagated in the crossed pyramidal fasciculus of the opposite side; if, on the contrary, there exist a focus in each of the two hemispheres, both halves of the pyramidal system may be simultaneously invaded.

And, I purposely repeat it, gentlemen, this has reference to a

systematic lesion *par excellence*. The adjoining fasciculi remain always indemnified, and (except under particular circumstances that we shall have to show) the gray substance and the roots of the peripheric nerves remain equally intact.

4. As to the nature of the degeneration, in an anatomical point of view, it is a question which I reserve. Is it the result of a purely passive process, as many authors claim, or, on the contrary, of an irritative one, as others pretend? Or rather should these two opinions be combined, in the sense, that while passive in the first period, the process becomes active in the second? Here are two interesting questions certainly, but, I repeat, we will consider them hereafter. For the present, I wish to confine myself to the topographical consideration of secondary degenerations, and you will see, as I have already stated, that this immediately concerns the systematized lesions in the domain of the pyramidal fasciculi.

IV. Let us take the secondary lesion at the period of its emergence from the depth of the encephalon. It is seen at first in the peduncle, thence we follow it to the protuberance, to the bulb, and finally into the several regions of the spinal cord.

Remark now, gentlemen, that if the lesion is very pronounced—one of long standing, it is very obvious. On the same side as the hemispheric lesion, you will observe a real atrophy of the inferior layer,—crusta, of the peduncle, of the protuberance and of the corresponding pyramid, which has, also, a grayish tint. On the side opposite to the lesion, below the decussation of the pyramids, you will also observe an asymmetry of the spinal cord due to the atrophy of the antero-lateral column. It is under this aspect that the lesion has been known by old writers. But observe, gentlemen, that in the great majority of cases, the changes do not proceed so far, and that, makes the intervention of the microscope indispensable.

1. In the peduncle, and this is an interesting fact, the degenerated part is greyish, and is seen in the form of a triangular space occupying the median division of the crusta. The base of the triangle is towards the encephalon, while the summit is towards the protuberance. Thus the surface of the crusta of the peduncle is, in a manner, divided into three regions; (*a*) a median region

represented by the degenerated pyramidal fasciculus; (*b*) an external region which never, under any condition (so for as I can judge, at least, after numerous observations which I have made on this subject), becomes the seat of degeneration,—a very remarkable circumstance on which I will dwell again; (*c*) finally, an internal region which, exceptionally, and under very special circumstances that I will also have to investigate hereafter, is invaded by the degeneration of the pyramidal fasciculus, or even may degenerate without participation of the median fasciculus.

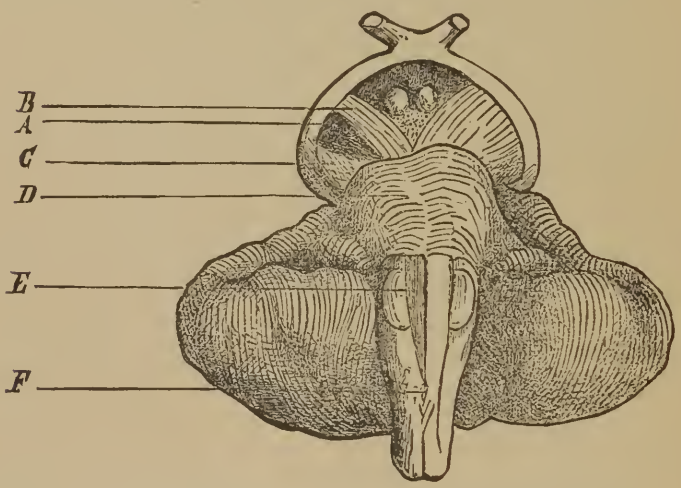

FIG. 12.—A degeneration in the cerebral peduncle of the pyramidal fasciculus. B, internal peduncular fasciculus; degenerates sometimes, but exceptionally. C, external fasciculus (centripetal ?), never degenerates. D, protuberance (pons v.); it is asymmetric and depressed on the side of the lesion. E, degenerated pyramid; grayish, atrophied, and decussating in F with the opposite side.

When we make thin sections of the peduncular region, we can assure ourselves, that the degenerated median fasciculus corresponds exactly to the pyramidal fasciculus, which the anatomy of development also confirms. With a low power, the lesion presents itself on a normal section under the form of a quadrilateral space occupying the median part of the crusta and extending from the gray substance of Sœmmering, which forms its superior limit and

is opposite to the narrowest side of the parallelogram, unto the surface of the crusta, which represents the longest side.

The nervous fibres being to a great extent destroyed in the area of the parallelogram, or replaced by the connective tissue, there is seen on carmine colored sections, a decided red tint in contrast with the relatively pale aspect of the neighboring parts, and this exhibits the exact topography of the lesion.

2. In the protuberance the disassociated pyramidal fasciculus is more difficult to recognize in the midst of the transverse fibres of that region. Nevertheless, the secondary degenerated fasciculi can still, plainly enough, be distinguished, especially by comparison

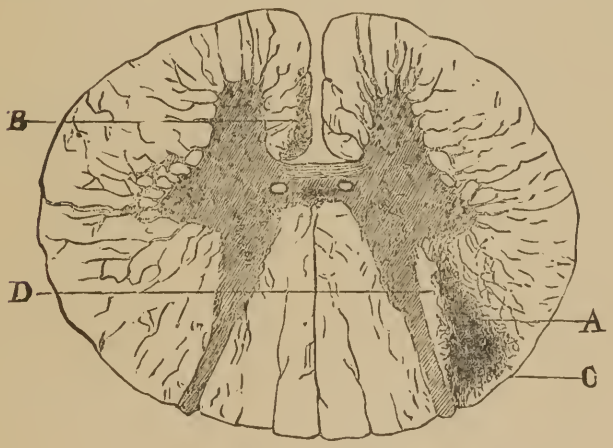

FIG. 13.—Transverse section of the cord in the cervical region. A, degeneration of the pyramidal fasciculus in a case of lesion of the hemisphere, motor centres. B, degeneration of the direct fasciculus. C, a space of white substance corresponding to the cerebellar fasciculus. D, region intermediary between the posterior horn and the pyramidal fasciculus; this region is always respected in descending degenerations.

with the healthy side in the inferior or bulbar region of the protuberance, where the secondary fasciculi begin to come together, and are rearranged in order to constitute, a little lower down, the pyramidal fasciculi.

3. In the whole length of the bulb, the lesion is readily recognized, which you may prove, by examining a section made perpendicularly

to the great axis of the rachidian bulb, on a level with the middle region of the olivary bodies.

The bulbar lesion is exactly circumscribed in the pyramidal fasciculus of the side of the primordial encephalic lesion.

4. If now we examine a series of transverse sections of the cord made in different regions, we will recognize at once the antero-lateral column of the side corresponding to the cerebral lesion; and on the side opposite to the lesion, the crossed pyramidal fasciculus. You see, gentlemen, how this last fasciculus may be followed separately as far as the inferior extremity of the lumbar cord, and how in the diverse spinal regions its connections are modified in accordance with the teachings derived from the anatomy of development.

The demonstration which I have aimed at is thus complete; and the descending degeneration is, as I have announced to you, literally embodied in the system of the pyramidal fasciculi.

V. After having determined the topography of so-called secondary degenerations, it is now necessary for me to pause and dwell on one of the most important points of their history.

What is the common character of all the destructive focal lesions, which on being developed in the depths of the hemisphere, give rise, after a certain time, to descending degenerations? On the contrary, what is the character of the lesions of the same order which do not produce degenerations? I repeat it, this concerns especially a question of seat, or, in other words, of localization, and certainly it is necessary to demonstrate it.

The cerebral hemisphere, I remarked to you at our last session, may, in a very general way, be considered as composed, up to a certain point, of two parts, anatomically, functionally and pathologically distinct; they are on one hand the central ganglionary masses, and, on the other, what is often called the mantle, that is the semi-oval white substance and the cortex of gray substance, which cover them.

1. Let us pause, in the first place, to consider the gray central masses. A very summary analysis of this region exhibits (on a transverse section), the white tract, called the internal capsule, with an antero-posterior direction; outside of this tract we see the

lenticular nucleus, and within the optic bed and the caudate nucleus.

It is not rare, indeed quite the contrary, gentlemen, to see destructive focal lesions, lesions narrowly localized, in one or the other of these nuclei. It is very common, so far as the lenticular nucleus is concerned, more especially in its external borders. There, are seated, you know, most frequently the ochreous foci—the last vestiges of intracephalic hæmorrhages. The foci, more or less numerous, which are limited to the optic bed, and those circumscribed in the head of the lenticular nucleus are not, either, very rare. Now, gentlemen, this point is established by Türck, who is the pioneer in the methodic study of secondary degenerations, that they never occur when the lesion, of whatever character, remains limited in the gray nucleus, and does not involve the internal capsule in such a manner as to alter, profoundly, the structures of its fibres.

The lesion of the internal capsule is therefore, so far as relates to foci in the central gray masses, the condition *sine qua non* for the production of secondary degenerations.

But, gentlemen, all of these destructive lesions in the internal capsule, do not give rise to secondary degenerations. We have seen that those which occupy a certain space, that may be estimated, at the least, as one-fifth of an inch in diameter, do positively determine them; while others, of much greater size, never produce them. What is the reason of these differences? All depends, as you will soon see, on the part, in the capsule, which is occupied by the lesion. The problem then is to define exactly the region of the capsule where a destructive lesion is followed by degeneration, in contradistinction to those where lesions do not produce the same result.

It is now four years since I formulated, on this subject, the following propositions: "Secondary degenerations are produced where the lesion bears on the anterior two-thirds of the internal capsule; they do not occur when the lesion occupies the posterior third of the internal capsule."

This proposition has been criticized by Flechsig, who has devoted himself profoundly to the study of this subject; which

has led me to review the facts on which I based my proposition, and I am led to recognize that on several points they are well founded. I have therefore modified my formula and will in a moment state to you in what consists the modification that I propose.

You will recall that the topographical study which we have made, has led us to recognize in the internal capsule, the existence of two segments, one anterior, the other posterior, and

FIG. 14.—Degeneration of the internal fasciculus of the peduncle (in an epileptic). D, the degenerated fasciculus occupies the internal fifth of the right peduncle. The protuberance (pons) and the bulb (medulla ob.) do not present any analogous lesion.

that these two segments unite at a point that Flechsig proposes to call the knee of capsule. Now, gentlemen, I have recognized that the lesions limited to the anterior segment determine a secondary degeneration; nevertheless this degeneration does not affect the pyramidal fasciculus; it is translated to the naked eye by the presence of a small gray band that occupies the internal

segment of the crusta of the peduncle, and does not affect the median segment. There exists, in all probability, interior to the pyramidal fasciculus, a fasciculus of centrifugal fibres proceeding from the anterior segment of the internal capsule. It is also very probable that it comes to an end, in some point of the protuberance; for when this fasciculus is degenerated it cannot be traced in the corresponding pyramid, and *a fortiori* it does not descend into the spinal cord. If I have misapprehended the isolated existence of this fasciculus, it is because the capsular lesions that I have examined, affected not only the anterior segment, but also the median segment of the capsule, and in that case the complex lesion of the capsule extended downwards through the bulb and into the spinal marrow.

The region of the capsule that interests us for the present, and of which we are anxious to determine the limits as soon as possible, we must seek in the posterior segment,—the lenticulo-optic, of the capsule. If you divide from before backward the posterior segment into three parts about equal, (in such a case we should not pretend to mathematical precision), the portion of the capsule which responds to the anterior two-thirds is, justly that, in which, after the observations of Flechsig, and my own recent ones, a destructive lesion even of limited extent, could not exist, without being followed by a descending degeneration of the corresponding pyramidal fasciculus. This might be called the *pyramidal region of the capsule*, since the nervous fibres which traverse it seem to be a direct emanation from the pyramidal fasciculi.

In regard to the other third of the posterior segment of the capsule, I will merely indicate in passing (for it is a point on which I shall have to recur) that this region appears to contain centripital fibres only, and which, in any case, do not seem to submit to descending degeneration. We always see that the local lesions developed in this point are translated, clinically, by a collection of symptoms, now well understood under the name of cerebral hemianæsthesia. This assemblage of symptoms is, I remind you, characterized as follows: (1) loss, or simple obnubilation of the general sensibility in the whole extent of the opposite side of the body; (2), on the same side the special senses, comprising the

vision and smell, are simultaneously affected. From these anatomico-clinical data, established now by a sufficient number of careful observations, the region in question may be considered as a sort of crossroads where are found united at all times in a narrow space, the conductors of general and special sensibility, which proceed from the opposite half of the body.

This fact is worthy of being brought alongside of that other fact, that in the crusta of the peduncle, at least according to my observations, otherwise quite numerous, the external segment is never touched by a secondary degeneration. Now the fibres of the external segment are those that Meynert, directed by pure anatomical considerations, considers to be centripetal fibres—a continuation of the sensitive spinal fibres and uniting them to the posterior regions of the cerebral hemisphere. The fibres of the external segment of the crusta remount then, directly, into the posterior segment of the capsule. This is only an hypothesis, doubtless, but it is one reasonable enough, and which is worthy, in ulterior researches, to be taken into consideration.

According to that view, the three regions of the capsule are represented in the crusta of the peduncle by the three fasciculi which have already been under consideration: (1), within, a fasciculus corresponding entirely to the anterior segment of the capsule, which seldom is degenerated; (2), a median fasciculus which responds to the anterior two-thirds of the posterior segment of the capsule,—the pyramidal fasciculus, whose degeneration is so common; (3), an external fasciculus which never degenerates, composed of centripetal fibres, destined to form the posterior third of the capsule.

I would not wish, gentlemen, to give you that as a definite result, but as a new view destined to serve as a guide in future researches.

Be that as it may, if I am not mistaken, one fact clearly arises from the preceding discussion, namely, that so far as the central grey masses are concerned, the destructive lesions affecting primarily or secondarily the anterior two-thirds of the posterior segment of the internal capsule, are the only ones which determine descending degenerations of the pyramidal fasciculi. Now this

region of the capsule is, precisely, that of which it is of moment to establish the circumscription; and, with this remark, I terminate the first part of my demonstration.

LECTURE FIVE.

SECONDARY DEGENERATIONS OF CEREBRAL ORIGIN, LIMITS OF THE PYRAMIDAL FASCICULUS IN THE CEREBRAL CORTEX.

Summary:—Secondary Degenerations Following Lesions of the Cortex. Median Ascending Frontal and Parietal Convolutions.—Pyramidal Giant Cells of the Cortex, and of the Spinal Gray Substance.—Pyramidal Giant Cells Found in Lower Animals in Same Regions.—Schematic View of the Rolandic or Motor Region of the Brain.—In the Cortex of this Region Destructive Focal Lesions Produce Secondary Degenerations of Pyramidal Tract.—Secondary Degenerations Connected with White Tract Beneath the Cortex, and in the Protuberance and Bulb.

GENTLEMEN:

It is now necessary to consider the cerebral cortex or mantle of the hemispheres, for there also, circumscribed or focal lesions originate secondary degenerations.

It was first announced by Türck, long ago, that peripheral focal lesions in contradistinction to those called central, because these occupy the opto-striated region, give rise also, under certain circumstances, to secondary degenerations resembling those which proceed from lesions of the internal capsule.

But under what circumstances do secondary degenerations result, when the lesions occupy the cerebral cortex?

It is only latterly that it has become known, that here, again, the fundamental condition is related to the *locality* of the focus. The extent of the primitive lesion, the nature even of the alteration that constitutes it, provided always that it is destructive, are in this species but accessory conditions. It is now important therefore for us to determine, topographically, in the cortex, the region where focal lesions would effect secondary degenerations of the pyramidal fasciculi.

I. In order to attain, surely, the end that we now propose, a slight digression, it seems to me, is indispensable. Besides, gen-

tlemen, in following this indirect route where I wish to conduct you, we shall disclose as we proceed many cerebral topographical facts that we shall be able frequently to utilize in the class of studies that we have undertaken.

When we examine the surface of the cerebral hemispheres in man, and seek to find there, well marked lines to guide us in that labryinth, which has always been called the cerebral convolutions, our attention is necessarily arrested by the remarkable disposition which the two great median convolutions present. They were first described and delineated by Vicq d'Azyr, in his great treatise on anatomy and physiology (1785, pl. 111). "*They are.*" says Vicq d'Azyr, "*directed obliquely from above downwards; they are more extended and less warped than in the other regions of the brain.*"

These two convolutions have been described with more detail by Rolando, in 1829, in a work entitled "*De la structure des hemispheres du cerveau.*" He designates them as the *processus enteroides verticaux*. In fact, gentlemen, these two convolutions constitute one of the fundamental morphological characters of the surface of the human brain; and they are found also in most monkeys. You know that now they are designated—the anterior, as the ascending frontal convolution, the posterior, as the ascending parietal convolution. They are separated from each other by a fissure that, since Leuret (1839), has been called the fissure of Rolando. On the external face of the hemispheres these fissures extend from the fissures of Sylvius, as far as the interhemispheric cleft; and it is said besides, that they are prolonged in a certain manner on the internal face of each hemisphere, and constitute a small lobule called paracentral or oval, at the level of which the two prolonged convolutions are united. The region of the gray cortex of the hemisphere, to which I have called your attention, is distinguished from all others, not only by its very peculiar configuration, but also by some details in structure which merit further attention.

Histological study, as you are aware, has demonstrated in the cortex of the hemisphere the existence of ganglionic or nervous cells, which, on account of their form, are called the *pyramidal cells* of the gray cortex. These cells vary greatly in size; some of them certainly are relatively, very small, and these are the most

numerous (pyramidal cells of the small species.) There are others more voluminous, which occupy the median plane of the gray substance. Finally, there exist pyramidal cells, called giant cells, which have been so carefully described by Betz and Mezierjewsky, the diameter of which is as great as 0.040 m. to 0.050 m., that is, equal to the diameter of the great ganglionic motor cells in the anterior cornua of the spinal cord.

But it is not only in respect to dimensions that there is ground to establish a relation between the cells of the anterior cornua of the cord and the great pyramidal cells of the cortex. There exists indeed, between these two elements, very manifest analogies of structure. Thus, besides the protoplasmic prolongations which ramify and subdivide so obviously, there is found in the large pyramidal cells, and particularly in the giant cells, a well characterized arrangement consisting of a special undivided prolongation, identical with the cylindrical prolongation, described by Deiters, in the spinal ganglionary cells. It is in both cases, at its origin, a delicate filament, which gradually enlarges in proportion to its separation from the cellular protoplasm. By this happy disassociation, it is possible to see this prolongation at a certain distance from the cells, receive its investment of a myeline cylinder.

All these explanations establish analogies, which show, notwithstanding the differences in form and seat, a close relation between the giant pyramidal cells of the cerebral cortex, and those of the motor cells of the anterior cornua. These analogies have, moreover, been presented already by Luys, and I shall have very soon, occasion to place them again, in relief.

Furthermore, the greatest of these pyramidal cells, and more especially, the giant cells, are not located indifferently in all parts of the cortex, but they are cantoned in a well defined portion of the cerebral surface, and in that, precisely, of which we have just indicated the configuration and boundaries. It is, in fact, in the depth of the gray substance of the ascending frontal and parietal convolutions, in their superior moiety principally, and in the paracentral lobule, that we find the pyramidal cells of the larger species, and exclusively, the giant cells, which are disposed there

in groups, in islets and in nests, as Betz says. Therefore the region of the median convolutions may be designated as the department of the gigantic pyramidal cells. It is very remarkable that this peculiarity of structure does not occur in man only. It is found also in the monkey, as Betz has shown. In them also, it is in the median convolutions and in the paracentral lobes that are seen the largest pyramidal cells. The same author has equally shown that these cells are found in the dog, in the regions designated, since the experiments of Hitzig and Ferrier, as the motor centres, that is, in the gray substance of the convolutions which border the *sulcus crucialis*.

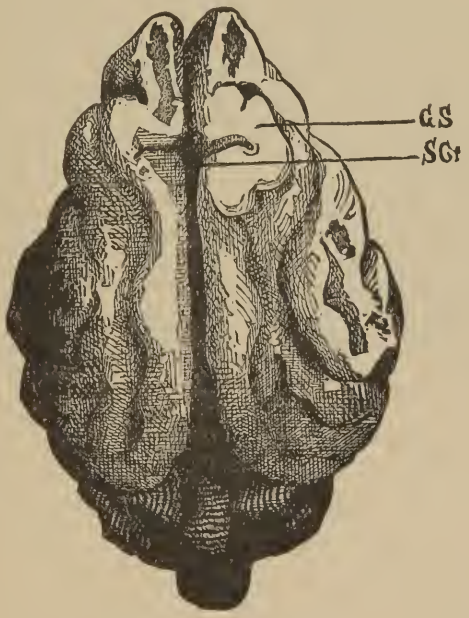

FIG. 15.—Brain of a dog (superior face). S.C.r., crucial sulcus. G.S., sigmoid gyrus (region of the convolutions with the large pyramidal cells).

The observations of Betz have recently been confirmed, by Bevan Lewees, not only in what concerns man and the monkey, but also in the cat and the sheep, (*Brain*, 1878).

These things lead me to declare that the central, median or Rolandic convolutions, if you will allow me thus to define them, are those in which experiment, in regard to the monkey and anatomico-clinical observation in man, have enabled us to localize the so-called psycho-motor regions, or more simply, motor; and I avail myself of this occasion to say to you, in passing, that the denomination, motor centres, does not implicate in my mind any physiological idea absolutely fixed; but, by that, I understand only, how to designate by contrast with others, those regions of the cortex of the brain, the lesion of which, occasions motor troubles in certain definite parts of the opposite side of the body.[1]

Here, gentlemen, our digression ceases. It may be that you have thought that these considerations, which I have presented to you, are foreign to our subject, which is the pathogenic history of secondary degenerations of cerebral origin; it is not so, however. Indeed that part of the gray cortex of which I have endeavored to show the principal morphological, histological and physiological characters appertains to a region, the limits of which I shall now attempt to determine. Proceeding from the lowest layer of the gray zone, the nervous fibres lead toward that portion of the internal capsule that we have heretofore named *pyramidal*, and you will obtain a schematic view, which is very near the concrete reality. Geometrically, this region may be represented as a pyramid of four faces. The truncated summit looks downward and covers the *pyramidal* region of the internal capsule. The convex base is directed upwards and outwards and includes the gray cortex of the median or ascending convolutions. The anterior face is represented by a frontal section passing on one side, through the posterior extremity of the base of the three frontal convolutions; on the other, through the anterior extremity of the optic bed (this is nearly the pre-Rolandic or pediculo frontal section of Pitres). The frontal section, post Rolandic or pediculo-frontal, passing on one side through the base of the parietal convolutions, and on the other through an imaginary line which separates the two anterior thirds of the posterior segment of the capsule from its posterior third, represents the posterior face of the

1 See for further details my lectures on Diseases of the Brain.

pyramid. It is easy to understand, without further explanations, how the two faces, internal and external, could be shown.

Here then we have enclosed within these pyramidal lines, what is known as the Rolandic region of the cerebral cortex, and which represents in a large, as in a small brain, a region endowed with special physiological properties; it is also in the cortex of this cerebral segment that we must search for the point

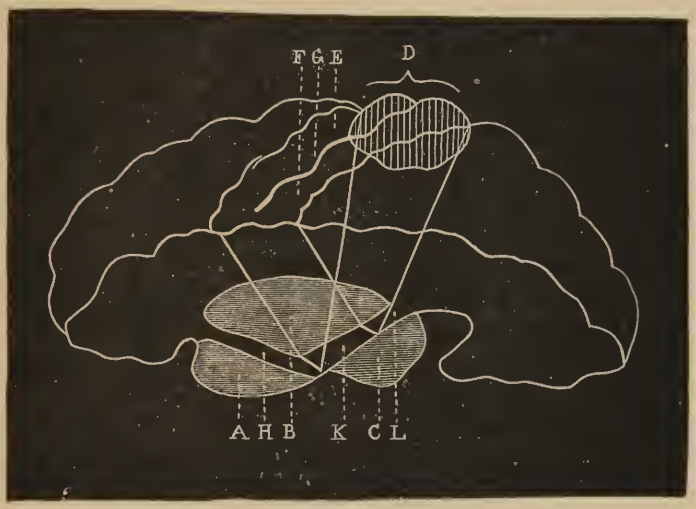

FIG. 16.— A, horizontal section of the nucleus caudatus. B, of the lenticular nucleus. C, of the thalamus opticus. D, paracentral lobule. E, frontal ascending convolution. F, parietal ascending. G, fissure of Rolando. H, anterior segment of the internal capsule. K, pyramidal region of the internal capsule in the posterior segment. L, sensitive portion of the capsule corresponding to the posterior third of the posterior segment.

of departure of secondary degenerations. Indeed so far as it concerns focal peripheric lesions, that is, those seated outside of the opto-striated masses, they do not determine secondary degenerations of the pyramidal fasciculi, except when they affect the central or Rolandic region of the hemispheric cortex. Outside of this region destructive focal lesions, whatever be their dimensions, do not produce descending degenerations of the pyramidal fasciculi.

Such is a summary, gentlemen, of the formula which expresses fairly the actual state of our knowledge on the subject. I shall not enter into a detail of the facts on which it rests. I refer you to the works of Pitres, Issartier and Flechsig, in which are collected all the documents relative to the matter. I will limit myself to the following remarks. It is not yet demonstrated that destructive lesions, limited to the gray cortical substance of the Rolandic region, produce secondary degenerations. Nevertheless some facts obtained from the pathological anatomy of general paralysis, seem to show that such is really the case.[1]

Lesions even of small extent (one to two centimetres) affecting at once the gray substance and the subjacent white substance, such as the yellow plates or superficial foci of cerebral hæmorrhage, more especially when they bear upon the two superior thirds of the ascending convolutions and the paracentral lobe, produce very decided degenerations of the pyramidal fasciculi.

Focal lesions affecting the *centrum ovale* in the Rolandic region, without participation of the gray cortex, determine by the same law as in the superficial lesions, secondary degenerations of the pyramidal fasciculus.[2]

To finish the particular history of secondary degenerations of cerebral origin, there remains only to say a word on what relates to foci limited to any of the regions of the isthmus. You will find embraced in the monograph of Bouchard, the greater part of the known facts in regard to consecutive secondary degenerations localized in the protuberance and the bulb. The comparison of these facts show, that the necessary condition, under like circumstances, for the production of secondary degenerations, is that the focus shall affect the tract of the pyramidal fasciculus. This being the case, a focus, even a very limited one, seated in the bulb at the level of the decussation, for example, would determine a descending degeneration of the two pyramidal fasciculi. This combination is found realized in an interesting observation published by Hertz.[3]

A focal softening, large as a lentil, seated at the point of decus-

1 The case of Déjerine, cited by Issartier.
2 Flechsig (Second Memoir four cases borrowed from Türck.
3 Deutsch. Archiv., 393, 1874.

sation produced a symmetrical secondary degeneration of the pyramidal fasciculi, which could be followed in the whole extent of the spinal cord.

LECTURE SIX.

SECONDARY DEGENERATIONS OF CEREBRAL ORIGIN. CONSECUTIVE AMYOTROPHY.

Summary:—Mode of Termination of the Fibres of the Pyramidal Fasciculi in the Spinal Cord.—Descending Degeneration of Pyramidal Tract arrested by the Motor Cells in the Anterior Horn of the Cord.—Muscular Atrophy in relation to Functional Inertia of Muscles, and to Destruction of Motor Cells in Anterior Cornua; Examples Furnished.—Protection Afforded by Anterior Cornua to Correspoding Anterior Root.—Exceptional Instances.

GENTLEMEN:

I would have finished the particular history of secondary degenerations of encephalic origin, if I had not felt it my duty to pause before proceeding further, to direct your attention, for a short time, to some facts in detail, which have not been given in my preceding remarks.

The anatomy of the pyramidal fasciculi derived from the study of the development of these tracts, and from the effect of their descending degeneration, permitted me, as you will remember, to establish that the nervous fibres which compose them, originate in the gray cortex of the Rolandic convolutions, and that from that point of departure, they descend as far as the cord without forming other relations, than those of contiguity, with the different parts of the encephalon and the bulb which they traverse. Now we can suppose, though indeed it may not be demonstrable, that in the gray cortex these nervous fibres are in relation, more or less directly, with the great pyramidal cells of that region; but below, in the spinal cord, where and how do these same fibres terminate?

We have seen that in the cord the pyramidal fasciculus grows gradually smaller in proportion as it descends to the *filum terminale*. This fact sufficiently proves that the nervous fibres that compose it, gradually disappear in the descending course of the fasciculus through the different spinal regions. On the other hand, certain

observations in normal anatomy show that in the different sections of the cord, there are fibres directed from the rear forwards, and from without inwards, which seem to establish a connection between the pyramidal fasciculi and the anterior horns of the gray substance.

In regard to the termination of these nervous fibres, which appear to be a direct emanation from the constituent fibres of the pyramidal fasciculi, several hypotheses have been presented, viz. : either the pyramidal fibres pass directly into the anterior roots, or they terminate in the gray horns, without going further, or, finally, it is possible that some of them are prolonged into the commissure and thus reach the opposite side of the cord.

Against the first hypothesis the following objections may be offered : The anatomy of the cord in the new-born shows, according to Flechsig, whose assertions on this point I can confirm, that while yet the development of the pyramidal fasciculi is barely traced, that of the anterior cells and the anterior roots is already greatly advanced. There is therefore no *continuity* between the pyramidal fasciculi and the fibres of the anterior roots. This fact finds, in some respects, its counterpart in the history of *descending degenerations*. In fact, when a lesion of one of the pyramidal fasciculi is most pronounced, it is the rule, that the anterior roots, emanating from the gray cornu of the corresponding side do not present any modification of structure nor any appreciable diminution of volume.

The gray substance itself of the anterior cornu, in the neighborhood of the degenerated pyramidal fasciculus, does not exhibit, either, in the ordinary conditions understand, any trace of alteration : thus the great nerve cells are perfectly intact, and the cornu offers no diminution in volume.

But, gentlemen, this does not prove that there exists no connection between the terminal extremities of the pyramidal fibres and the multipolar motor cells; indeed, it is very reasonable—and this is the opinion of a majority of writers, that such a connection does exist.

The second hypothesis, which I have formulated above, would, by this view, be truly confirmed; nevertheless, you should know,

that the anatomical arrangement, according to which this relationship is established, up to this time, remains undemonstrated.

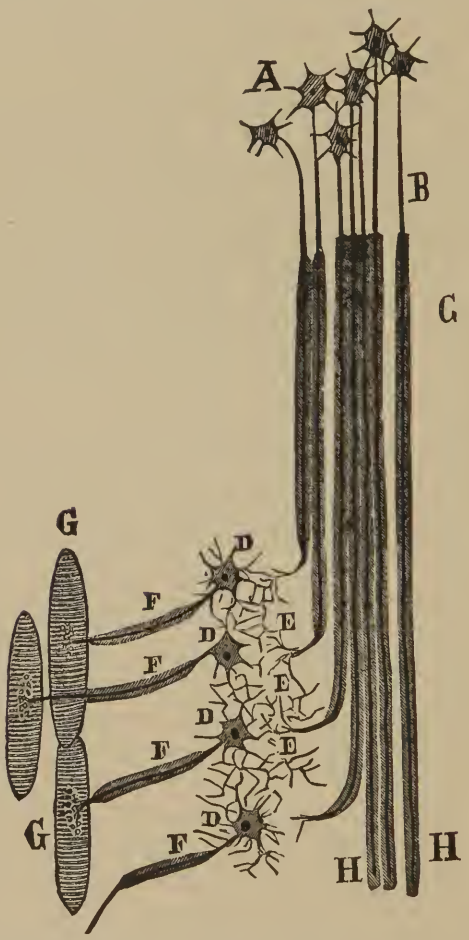

FIG. 17.—Schema indicating the whole of the apparatus of the pyramidal fasciculus from the cells in the gray cortex of the brain unto the terminal plates in the muscular fibres. A, giant cells of the so-called *motor* convolutions. B, axes cylinders. C, axes covered with myeline, forming the pyramidal fasciculus in the lateral columns of the spinal cord. D.D.D., cells of the anterior horns of the cord. E.E.E., reticulum of the gray substance where disappear successively, the nervous fibres of the lateral column. F.F.F., anterior roots represented schematically, by the axes cylinders which proceed from the anterior cells, and terminate in the muscular fibres G.G.

However that may be, the nervous motor cell, in the case of descending degeneration, should be considered as the obstacle which arrests, in the gray substance, the work of degeneration and prevents its propagation to the nervous tubes of the anterior roots, which physiology, unquestionably, shows have mediate relations with the fibres of the pyramidal fasciculi. This hypothesis, as you can judge, finds a support in certain pathological facts appertaining to the history of descending degenerations, to which I wish to direct your attention for a short time.

It has long been observed that, as a rule, the muscles of the paralyzed side in patients attacked with permanent hemiplegia of cerebral origin, do not present other atrophic alterations than those which, very tardily, result from the functional inertia to which they are condemned. But in this regard there exists still a chapter of exceptions, and it may happen, contrary to the rule, that the muscles of the stricken members in hemiplegia, submit at a given time, to an atrophy more or less rapid, while, at the same time, they manifest more or less profound modifications in their electrical reaction. I have been led to think that this anomaly, properly belongs to some anatomico-pathological peculiarity, and, in one case, I have indeed established the following facts:[1]

They relate to a woman attacked with left hemiplegia, subsequent to a focal hemorrhage in the right hemisphere. The members of the paralysed side, which were seized, very early, with contracture, began to diminish in size, two months after the attack. The muscular atrophy was uniformly spread in all portions of the paralyzed members, and was accompanied with a notable diminution of electrical contractility. The muscular atrophy progressed very rapidly—the patient died, and at the autopsy we recognized, in hardened sections of the spinal cord, that, besides the sclerosed fasciculus, there existed, in the anterior horn of the corresponding side, an alteration, of which the most salient point was their atrophy, and, even the complete disappearance of a certain number of the motor cells.

Hallopeau, while in the service of Vulpian, observed similar facts. Leyden has likewise seen one similar case, though he has

1 See Lectures on Diseases of the Nervous System, T. I, p. 35, and T. II, p. 245.

not, I think so at least, shown the relation between the alteration of the nerve cells and the atrophy of the muscles.

Quite recently, Pitres,[1] concerning a fact of the species that we are now considering (observed in my service four years ago), has given very important anatomico-pathological details. It related to a case of yellow focus, the remains of an old cerebral hemorrhage. The focus—about the size of a large almond—had totally divided the middle third of the internal capsule of the right side. During life the muscular atrophy, which had attracted attention occupied nearly the whole of the left superior member. On the contrary, the muscles of the left inferior member presented no notable atrophy.

Enlightened by the preceding facts, I felt justified in announcing

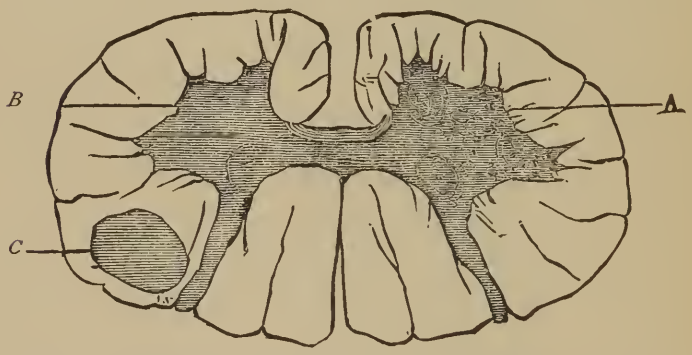

FIG. 18.—Section of the spinal cord between the seventh and ninth rachidian pairs. A, anterior horn of the right side containing motor cells perfectly sound and in normal number. B, left anterior horn ; the motor cells have disappeared save on the most external and on the antero internal parts. C, sclerosis of the pyramidal fasciculus in the lateral column (after Pitres).

that this isolated muscular atrophy arising in the progress of a cerebral hemiplegia with secondary contracture would find its explanation in a partial destruction of the cells of the left anterior cornu, on a level with the cervico-brachial region. The microscopical examination, made with the greatest care by Pitres, fully confirmed my predictions.

1 Archives of Normal and Pathological Physiology, 1876, p. 664.

The descending sclerosis existed on the left side of the whole length of the cord; and, besides, in a limited point of the cervico-brachial enlargement, between the seventh and ninth pairs of spinal nerves, and to an extent of 2 to 3 centimetres, there existed in the cornu of the anterior region, a complete disappearance of most of the cellular groups which are found normally, in that region (posterior, and antero-external groups). Moreover, the anterior roots in the region corresponding to the cervico-brachial enlargement were grayer than in symmetrical points of the right side. Facts of this nature form a group sufficiently homogenous, to show, that it is not a mere fortuitous coincidence.

One of the lessons which they furnish is, that the terminal exremities of the pyramidal fibres, as we have said, are in relation, in some manner, with the nerve cells of the anterior cornua. As a rule when there exists a secondary degeneration of the lateral cord, the cells resist, by reason of their autonomy, the invasion of the morbid process and protect, so to speak, the corresponding anterior roots. But in certain exceptional cases, and probably, less rarely than is commonly supposed, under the influence of certain inexplicable conditions the cells are attacked also,—become atrophied, and, consecutively, the corresponding roots submit to a degenerative alteration. The final result of this invasion of the centrifugal nerves is the atrophy and degeneration of the muscles to which the diseased roots are related, following a mechanism which we shall study in detail on a future occasion.[1]

It is important for this theory, to remark, that in the case of Pitres, as in those which have preceded it, the descending degeneration of the pyramidal fasciculus presented no anomaly in its arrangement, in the neighborhood of the altered cornu. The islet of degeneration was as usual separated from the gray substance by a white tract; in other terms, there could not be found any sign of a direct extension of the lesion of the lateral fasciculus into the corresponding anterior horn.

That observation comes, then, to the support of the idea, that

1 Since this lecture was delivered (April 21, 1879) new cases of muscular atrophy consecutive to secondary degenerations of cerebral origin, have been observed in the service of M. Charcot. The complete study of these observations has been published recently, in the *Revue Mensuelle de Medicine et de Chirurgie*, August, 1879. E.B.

the propagation is not made by the intermediation of the connective tissue, but, by following, exactly, the tract and by way of the nervous fibres which, parting from the pyramidal fasciculus gain the horns of the anterior gray substance. Anatomy indicates that this relation is established by the aid of small fasciculi of nerve fibres, which, from point to point, are detached from the pyramidal fasciculus.

An attentive study will yet enable us, possibly, in cases of descending degeneration where the fibres are affected, to distinguish them amidst the sound fibres in the region of the reticular process. This very difficult observation has not yet been made.

Gentlemen, we have entered into details, which may appear to you very minute. but assuredly, you will not have to regret it, for we come, if you will allow the expression, to lay our hands on data of the first order, and which we shall utilize very soon for the physiological interpretation of amyotrophies of spinal origin.

LECTURE SEVEN.

SECONDARY DEGENERATIONS OF SPINAL ORIGIN. ACENDING DEGENERATION OF THE CEREBELLAR FASCICULUS, AND DESCENDING OF THE PYRAMIDAL FASCICULUS.

Summary:—Systematic Lesions from a Focal Destructive Lesion.—Compression of Spinal Marrow from a Pachymeningitis Externa Potts' Disease.—Compression from External Tumors.—Internal Tumors.—Total Transverse Lesion.—Ascending and Descending Degenerations which follow it.—The Pyramidal Fasciculi are incapable of an Ascending, and the Posterior Fasciculi of a Descending Degeneration.—Ascending and Descending Degenerations only follow a Destructive Lesion in White Fascieuli of the Cord.—Limitations in Destructive Lesions of Gray Substance.—Effects following Lesions in Different Fasciculi of the Cord.—Examples given.

GENTLEMEN:

Up to this time, we have been occupied, exclusively, with the pyramidal fasciculus; but we have only considered it in regard to normal and pathological anatomy. The symptomatology is no less interesting and we pause at this, equally. But, beforehand, I think it useful to make a summary of all medullary degenerations from a spinal cause, having in view still, however, as the principal object, the pyramidal fasciculi, the history of which must continue to occupy us, particularly, for this year.

This route, slightly deviated, apparently, must, nevertheless, lead us to the symptoms; and you will thus be more apt to appreciate their value, in the semeiology of spinal affections.

We proceed then to occupy ourselves with systematic lesions, of which I have already spoken so frequently,—fasciculated lesions, both ascending and descending, which are developed in certain regions of the cord as the consequence of the formation of a *focal destructive lesion.*

These secondary lesions of spinal origin are very common; for by reason of the relatively narrow dimensions of the spinal cord,

there can hardly exist a destructive lesion of that nervous column without secondary degeneration, under some form, following it.

I. Considering, to begin, a focal lesion as the cause of all the accidents, and we have a very simple case, and at the same time of a very common species. Let us take the slow compression of the spinal marrow, such as is seen, so commomly, in Potts' disease.

We know very well since the researches of Michaud, the ordinary mechanism of that sort of compression. The agent in such a case is the thickened dura mater. On its external surface it is in contact with the caseo-tuberculous deposit which issues from the vertebral bodies, and when the destruction of the posterior vertebral ligament is complete there arises an inflammation equally caseo-tuberculous (*pachymeningite externe caseo-tuberculeuse*). On account of this thickening of the dura mater, the marrow is repelled and compressed to an extent that varies according to the development of the caseous neoplasm, generally as long as two or three centimetres. Sometimes it is only compressed from before backward, at other times it is squeezed on all sides—in some sort strangulated.

But the action there is not always a pure mechanical phenomenon; in fact, there is soon produced in the organ, in place of compression, a reaction of an inflammatory order; in other terms, a partial myelitis is a consequence—nearly obligatory, from the spinal compression. Be that as it may, there is developed there a destructive lesion which, in the region lesed, affects indiscriminately the gray substance and all of the white fasciculi, both anterior and posterior; it is what may be called a *total transverse lesion*. It is well to understand that this same alteration is produced outside of Potts' disease, with the same characters and the same effects so far as it concerns secondary degenerations, whatever may be the cause of the compression. Thus cancerous and sarcomatous tumors, psammoma, originating without the spinal marrow, are sufficient as they grow, to determine a transverse myelitis by compression, with all its consequences.

Moreover, gentlemen, compression, properly speaking, is not a necessary fact. Intra-spinal tumors, glioma, syphiloma, solitary tubercles, syphilitic, traumatic, or other forms of myelitis will be

followed by the same conditions as in myelitis from compression, provided that it fulfil the express condition,—destruction of the nervous elements.

Thus, from the moment a total transverse lesion acts as a destructive process, whatever may be its origin, the result is always the same. The only varieties are in relation to the seat; therefore, degenerative lesions present certain peculiarities depending on the initial lesion occupying one region or another of the spinal cord. It is the same thing in a case where the transverse lesion is partial, that is to say, when it infringes only on a part of the dimensions of the organ. But, as you will recognize, the general law which dominates the development of secondary degenerations of spinal origin, is not sensibly modified in such a case.

II. For greater simplicity, we will consider, first, the case of a total transverse lesion, and we will suppose that it is seated in the superior dorsal region.

We must now take into consideration the descending degenerative lesions radiating below the lesion, and the ascending, radiating above it.

1. *Descending Secondary Degenerations.*—(*a*) Immediately below the focus, to an extent of one or two centimetres at most, the whole area of the antero-lateral columns is altered; only the posterior columns remain indemnified.

(*b*) Lower down observe what has taken place: two little bands of sclerosis in the antero-lateral fasciculi represent a degenerative lesion; these two bands are the direct pyramidal fasciculi or the columns of Türck.

A. In the lateral portion proper, of the antero-lateral fasciculi, the degeneration occupies the two crossed pyramidal fasciculi only. But the columns of Türck rapidly disappear, unless anomalous, and the pyramidal fasciculi then, alone, present the character of descending degeneration. Here, the same as in degenerations from a cerebral cause, except in certain complications, the gray substance and the anterior roots remain indemnified.

2. *Ascending Degenerations.*—Ascending degenerations bear on two points: (*a*) the lateral fasciculi; (*b*) the posterior fasciculi.

(*a*) The lesion of the antero-lateral fasciculi has nothing in common with the descending lesion. The *pyramidal fasciculi* are here absolutely indemnified. They are not capable of degeneration upwards, while the posterior fasciculi are not capable of downward degeneration.

The lesion is seen on a section in the form of a thin band, which begins on a level with the posterior extremity of the corresponding posterior gray cornu, and extends forward into the neighborhood of a transverse line which would pass by the anterior extremities of the anterior horns and sometimes beyond that.

It is a very thin band, and by its external border, is everywhere in contact with the pia mater. This lesion, already known

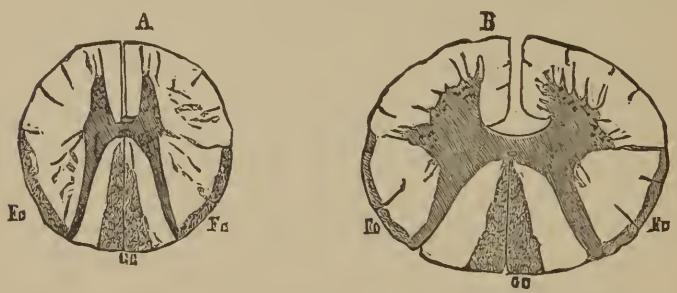

FIG. 19.—A, section of the spinal cord (superior dorsal region). B, cervical region. F.c., cerebellar fasciculus degenerated above the spinal lesion. G.C., columns of Goll.

by Türck, may be followed as far as the uppermost regions of the cord. It is found in the restiform bodies as high up as the level of the cerebellum.

The fasciculus which submits to this ascending degeneration does not seem to be equally developed in all subjects. The fibres which compose it appear to originate in the highest regions of the dorsal portion of the cord. Consequently, such a degeneration is not seen, if the focal lesion is seated in the inferior part of the dorsal region, or below that. The fasciculus in question designated by Flechsig as the *direct cerebellar fasciculus*, augments sensibly, in size from below upward. Flechsig supposes that the fibres arise from the vesicular column of Clark, but its anatomical connections are, thus far, quite obscure. I have nothing more to

say to you about this fasciculus of which the ascending lesion, so far as we know, is not translated into any special symptom.

(*b*) The ascending lesions of the posterior fasciculi are of greater interest.

A. Immediately above the focus, to the extent of two or three centimetres at most, the posterior fasciculus is lesed in its whole breadth.

B. But, higher up, the lesion seems to thin out, and occupies but little more than the median part, which in the superior regions of the spinal cord (cervico-brachial enlargement) corresponds to the fasciculus of Goll. (See fig. 19 G.C.) The degeneration may, moreover, like the fasciculus itself, be followed upward in the posterior pyramids of the bulb, unto the level of the fourth ventricle, while the lesion of the rest of the fasciculus does not ascend above the focal point to a distance surpassing the origin of two or three nerves.

Moreover, gentlemen, I shall later, enter into some developments relative to this alteration of the posterior fasciculi, which we now encounter for the first time, and which we are bound to study in still more detail, when we shall have to recur to locomotor ataxia.

III. At present, it remains for me to indicate briefly some modifications that the type presents which we have just described, when the lesion in place of being totally transverse, is but partially so.

(*a*) 1. First, it is important to show that degenerations, as well ascending as descending, do not exist except the destructive lesion attacks the white fasciculi. Thus, in even profound alterations of the spinal cord, when they are limited to the gray substance, as is always seen, either in an acute form, as in infantile paralysis, or in a chronic form as in protopathic spinal amyotrophy, they are never followed by descending degenerations unless it be by an accidental propagation to the white fasciculi.

2. In the white fasciculi, on the contrary, the consecutive lesion is in a manner, obligatory; if the primitive lesion is in the domain of the antero-lateral columns, and if it respects the pyramidal fasciculi, that degeneration will be of small extent, for the alteration affects in such a case only the very short commissural

fibres. We shall see presently what comes to pass when the focal lesion interests, uniquely, the fibres of the posterior fasciculi.

(*b*) Now, we must, for an instant, consider a combination which is encountered sufficiently often in practice, that is, a transverse, and unilateral lesion of the spinal cord. It is rare that the lesion is mathematically unilateral, that is to say, that it does not pass somewhat the antero-posterior axis; but in lieu of dealing in generalities, let us take a concrete example.

It relates to a syphilitic lesion of the cord which was translated into symptoms of spinal hemiparaplegia (on one side paralysis and hyperæsthesia; on the other, absence of motor paralysis, but anæsthesia)[1]. The autopsy demonstrated that the destructive lesion interested the totality of the left antero-lateral fasciculus and the totality of the posterior fasciculi. Above the focus, exactly circumscribed in the regions which we have just indicated, there was established a secondary degeneration of the two fasciculi of Goll; and below these was seen in the area of the antero-lateral column, the triangular degeneration of the left pyramidal fasciculus. It is in this way, bear in mind, that things are generally brought about in what concerns the pyramidal fasciculus.

But here is an exception to the rule, which it is not inutile to consider, because it will explain to you, perhaps, some symptoms which would otherwise be somewhat obscure. I have occasionally seen the two lateral fasciculi affected by a unilateral lesion. The alteration, it is true, was not symmetrical and eqnal; that of the side primitively attacked was more developed than the other. This disposition is seen in the most remarkable manner in the case of a traumatic section of the spinal cord observed by W. Muller.

It relates to a man aged 21, who received a cut with a knife at the level of the fourth dorsal vertebra. The cord at this point was cleaved in the whole of the tract of the right antero-lateral column, and in the posterior columns. (See fig. 20). This man presented the following symptoms: on the left side anæsthesia and conservation of movements; on the right, hyperæsthesia and paralysis of the leg. Death took place in about forty-three days, and the autopsy showed a descending degeneration occupying, not only

1 See Lectures on Diseases of the Nervous System, vol. ii.

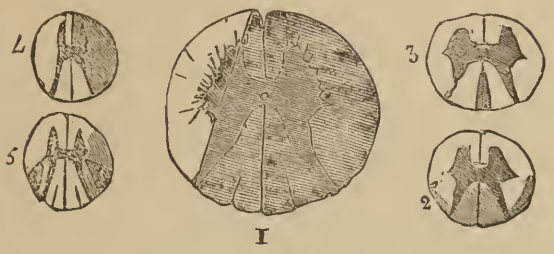

FIG. 20.—1, transverse section of the cord on the level of the fourth dorsal vertebra. The whole of the right half of the cord, and all the extent of the posterior columns are destroyed. 2, section of the cord on the level of the third dorsal vertebra (sclerosis of the two posterior columns and the two cerebellar fasciculi). 3, above, the degeneration occupies no more than the two columns of Goll. 4, the cord at the level of the sixth dorsal (degeneration of all of the antero-lateral columns). 5, lower down at the heighth of the seventh and eighth dorsal vertebræ the degeneration of the right lateral fasciculus is accompanied by a degeneration of the left pyramidal fasciculus.

the right antero-lateral column in its totality, but also the left pyramidal fasciculus.

I have observed facts of the same nature; Hallopeau has equally signalized them in certain cases of secondary degeneration of cerebral origin. This propagation of a primitive unilateral alteration to the opposite side can be explained, gentlemen, by the hypothesis which I presented at our last conference in respect to the mode of termination of the fibres of the pyramidal fasciculus in the gray substance. I said that the most of the fibres stopped in the anterior horns where they are placed in connection with the cells of the anterior horns. But it is possible that some of them pass into the anterior commissure, more especially in the dorsal region, and gain thus the lateral fasciculus of the opposite side to descend with it into the lumbar region. There would exist, then, for these fibres a double intercrossing—one in the bulb (anterior pyramid), and the other in diverse points disseminated in the whole length of the dorsal region; and if I insist on this peculiarity, it is because it is possible in this way to explain a paraplegia more or less complete, and paralyses of both inferior members, which are sometimes produced in the cases of unilateral spinal lesions, or from focal cerebral lesions.

LECTURE EIGHT.

ASCENDING DEGENERATIONS OF SPINAL ORIGIN. LESIONS OF THE FASCICULI OF GOLL, AND FASCICULI OF BURDACH. SPINAL DEGENERATIONS OF PERIPHERAL ORIGIN.

Summary:—Two Constituent Systems in the Posterior Fasciculi of Spinal Cord.—They are perfectly distinct Anatomically and Functionally.—Development of Posterior Fasciculi of Goll and Burdach as seen in the Embryo; these two Fasciculi may suffer distinct Degeneration.—Effects of Destruction of these Fasciculi from Compression.—Lesions in Locomotor Ataxia.—Secondary Degenerations of Peripheric Origin.—Lesion in the Cauda equina.—Results in cases that are recorded.

GENTLEMEN:

As I announced to you at our last reunion, we shall proceed to-day to review certain important facts relative to ascending degenerations of a spinal origin. I especially refer to lesions of the posterior fasciculi. But, beforehand, I must dwell on some particulars in regard to the normal anatomy of these fasciculi.

In the first place we must recognize that in the whole length of the spinal cord the posterior fasciculi, according to descriptive anatomy, constitute but one system alone; on the contrary, they must be decomposed into two secondary fasciculi, representing, in a manner, two systems—two organs perfectly distinct anatomically, and which exhibit separate functions both in the normal and the pathological state.

Now this autonomy of the two constituent systems of the posterior fasciculi is based on considerations derived first, from the anatomy of development; second, from the anatomy of structure; third, and finally, from pathological anatomy. We shall successively pass in review the three orders of facts on which is founded the distinction in question.

I hasten to remind you that the developments which are to follow will be for a great part only an exposition of the very important researches published by Pierret in the Archives of Physiology in 1872-3, at which time he did me the honor to labor under my direction.

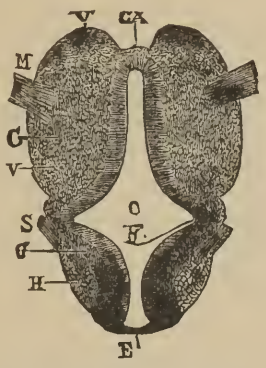

FIG. 21.—Transverse section of the cervical cord of an embryo of six weeks, 50 diameters. C, central canal. E, epithelial investment of the canal. G, anterior gray substance with a somber nucleus, whence emenate the anterior roots. G, posterior gray substance. H, posterior column. C.A., anterior commissure. M, anterior root. S, posterior root. V, posterior part of the anterior (or lateral column). E, thin portion of nervous substance closing central canal (Kolliker).

FIG. 22.—Section of the cord of a human embryo of one month. A,′ anterior horns. B,′ posterior horns. C, central canal. D, anterior roots. E, posterior roots. A, anterior radicular zone. B, posterior radicular zone.

I. A. A word, beforehand, about the development of the posterior fasciculi.

(*a*) In the human embryo of six weeks the posterior fasciculi

are represented by two little bands which cover, in a manner, the posterior cornua.[1]

They are the rudiments of the fasciculi of Burdach; the median fasciculi or fasciculi of Goll, are not yet developed.

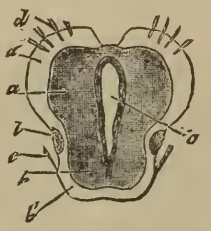

FIG. 23.—Section of cord of a human embryo of six weeks. The letters A.B.C., *et cet.*, indicate as above. L, lateral column.

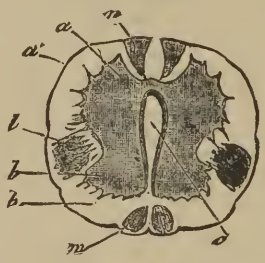

FIG. 24.—Section of an embryonic cord of two months, A.B.C., *et cet.*, as above. L, lateral fasciculus. M, development of fasciculi of Goll. N, development of fasciculi of Türck (anterior fasciculi).

(*b*) The latter begin to appear toward the eighth week, in the form of two buds, which seem to emanate from the fasciculi of Burdach. About the tenth week they become entirely distinct from them and are seen in the whole length of the cord.

(*c*) In the progress of development, the fasciculi of Goll, in certain regions, are fused with the fasciculi of Burdach without in every case, becoming confounded with them. But in the cervical region they remain distinct, even in a macroscopic point of view,

1 We reproduce here for comparison the demischematic figures, published in Charcot's Lectures on Symmetrical Scleroses of the Lateral Columns (after the preparations of Pierret).

and are limited on each side by the intermediary posterior furrows (Sappey). So much for the anatomy of development.

B. In regard to the normal or structural anatomy of this portion of the cord in an adult, the examination of hardened

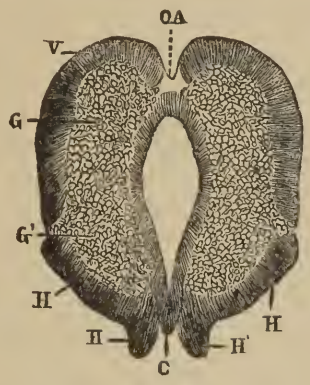

FIG. 25.—Transverse section of an embryonic cord of eight weeks (Kolliker.) H.′H.′ salient part of the posterior columns H. H., which later form the columns of Goll. C.A., anterior commissure. V, anterior column. G, gray anterior substance. G,′ gray posterior substance. H, posterior column. Between the two projections H. H., of the posterior columns, C represents the epithelium of the central canal (after Koiliker).

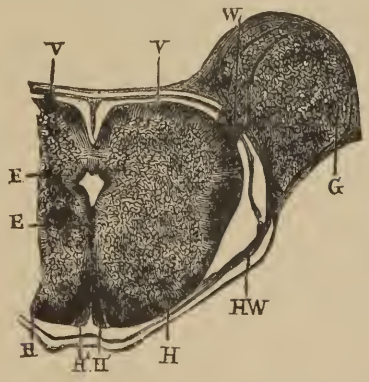

FIG. 26.—Transverse section of the cervical cord of an embryo of 9 to 10 weeks (35 diam). F, epithelium of the central canal. E, obliteration of this canal, posteriorly. V.V., anterior columns. H.H., posterior columns. H.′H.′ columns of Goll. V.W., anterior roots. H.W., posterior roots. G., intervertebral ganglion (Kolliker).

thin sections made in various directions shows: (*a*) that the fasciculi of Goll are composed of parallel fibres of long range, which, as commissures, bring into relation very distant levels of the central gray substance. Above, this system of commissural fibres terminates in a ganglionary mass which is seen on the floor of the fourth ventricle and is known as the *nucleus of the fasciculi of Goll.* Remark though, that the fasciculi of Goll have no connection with the intra-spinal prolongation of the posterior roots. (*b*) It is not the same with the fasciculi of Burdach which, on the contrary, are traversed by a part of the fibres emanating from the posterior roots. Some of the fibres of these roots, at the point where they join the cord, plunge directly into the gray substance of the posterior cornua; others do not attain the gray substance until they reach the neck (*cervix cornu posterioris*), after having traversed, following a much longer path, the fasciculi of Burdach where they describe a curve with an external convexity.

(*c*) But this group of radicular fibres emanating from the posterior roots does not constitute alone, by any means, the totality of the fasciculi of Burdach. The great mass of these fasciculi is formed, on the contrary, by vertical, arciform fibres very much shorter than the fibres of the fasciculus of Goll, and which are insinuated in very varied directions.

Such is, according to a direct study of the arrangement of the nervous fibres, the particular construction of the two fasciculi of the posterior column.

C. You have just seen, gentlemen, that the anatomy of development and the anatomy of structure concur to demonstrate the mutual independence of these two fasciculi. Now here is a new verification, which is furnished by pathological anatomy.

(*a*) The fasciculus of Goll, in certain cases, is seen systematically lesed in its whole extent, without any participation of the fasciculi of Burdach. This has been demonstrated by Pierret, from an observation made in my service. The same fact has been encountered since by other observers, and in each case the fasciculus is clearly designated in the whole extent of the spinal cord. In the lumbar region it is seen, in transverse sections, in

the shape of two little nodules, circumscribed in all parts by the fibres of the fasciculi of Burdach. These two fasciculi have, indeed, in that region, a major importance, by reason, more especially, of the great number of posterior radicular fibres which they receive. In the dorsal region, on the contrary, the median fasciculi encroach by their dimensions on the fasciculi of Burdach. Lastly, in the cervical region the fasciculi of Burdach become again enlarged, but the fasciculi of Goll are not on that account less voluminous; and it is quite important to remark, that it is in this region that they are, most distinctly, isolated from the adjoining parts, owing to the greater depth of the two intermediary posterior furrows. I must still remind you, in passing, that the symptoms observed in the cases of isolated systematic lesions of the fasciculi of Goll are not those of progressive locomotor ataxia.

(*b*) That which I have just presented, in respect to the fasciculi of Goll, is reproduced equally in that which relates to the fasciculi of Burdach. These may also submit to an isolate alteration, autonomic in some sort, without any participation of the fasciculi of Goll. Thus, in a subject who has presented all the symptoms of locomotor ataxia, as well in the inferior as in the superior members, the fasciculi of Burdach were alone lesed, and in the whole length of the spinal cord. As regards the fasciculi of Goll they were absolutely indemnified; the same fact has been established since in many observations.

C. Now, gentlemen, we are in a measure ready to comprehend the singular disposition that the lesion of the posterior fasciculi presents when it results from compression determined by a tumor which has produced the destruction, in one part of their tract, of the fibres of the fasciculi of Goll, and those of the fasciculi of Burdach. The lesion affecting the short commissural fibres of the fasciculus of Burdach determines upwards a short tract degeneration; and we shall soon see why this degeneration is ascending. Finally, the same lesion, insomuch as it bears upon the long commissural fibres of the fasciculi of Goll, determines equally an ascending degeneration which may be followed in the whole extent of the spinal cord as far as the floor of the fourth ventricle.

(*d*) If now, instead of a case of compression, you will imagine

one of partial transverse myelitis and limited to the posterior fasciculus, the result will be absolutely the same, at the point where the continuity of the fibres shall have been interrupted. Apposite to this, I wish to say, that in locomotor ataxia, the most habitual disposition of the lesions recalls with some slight modification what I have just said on the subject of ascending degenerations. The fundamental lesion involves, at its origin, the radicular fasciculi in the dorso-lumbar region; but as it relates there especially to an inflammatory process, the lesion does not remain confined in the focus of origin; it extends, step by step, in all directions, one part following the tract of the nervous fibres, another the connective tissue, and gains in this way on one side the posterior cornu, and on the other the fasciculi of Goll. The consequence of this propagation is, that the fibres of the fasciculi of Goll, being attacked in the inferior portion of their tract, degenerate from below upwards the same as if it concerned a direct compression, and the lesion thus produced may be followed, as has just been said, as far as the floor of the fourth ventricle.

A

FIG. 27.—A total sclerosis of posterior columns of Goll, and radicular zones—Burdach's (common locomotor ataxia).

B

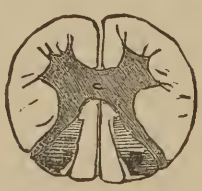

FIG. 28.—B., sclerosis of the two radicular zones—Burdach's (the fasciculi of Goll are untouched). Locomotor ataxia.

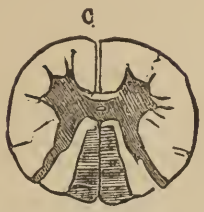

FIG. 29.—C., sclerosis limited to the fasciculi of Goll (ascending degeneration).

Such is, gentlemen, the common lesion of locomotor ataxia—that which is encountered ninety times in one hundred in those who succumb to this disease.

II. It is in accordance with the rule that prevails in common locomotor ataxia, that we must interpret the secondary degenerations, that constitute the third group, which it now remains for us to consider. By the side of secondary degenerations of cerebral and of spinal origin, the descriptive study of which is now ended, it is necessary to place the group of secondary lesions of peripheric origin.

This group is, thus far, composed of three or four observations only, but it is probable that the number will be increased. I shall limit myself on this point to citing for an example the observation of Cornil, the first in date, and one observation of Th. Simon.[1]

In all the cases observed up to this time the lesion is seated without the cord, on the roots of the cauda equina, above the ganglion; it consists of a sarcomatous or myxomatous tumor which invests and compresses the nervous fasciculi. It cannot be questioned that the spinal lesion in the observations which I have cited, arises, not from the anterior, but from the posterior roots. The consecutive degeneration is characterized, then, as follows: 1. It occupies exclusively the posterior fasciculi; 2. In the lumbar region the posterior fasciculus is invaded *totally in its transverse extent;* 3. But above this region the fasciculus of Goll is alone affected, and it is *totally in its whole length*, that is to say, as far as the floor of the fourth ventricle. Thus, you see, as

1 Arch. f. Psych. v. Westphal, Bd. 1874, p. 114.

regards topography, a sufficiently exact reproduction of the lesions of ordinary ataxia.

Now observe how these things are brought about, at least theoretically, according to the principles that we have hitherto formulated. Conformably to the law of Waller, the posterior roots degenerate upwards, above the lesion on the part of the cord, and the degeneration of these fibres may be followed in their intra-spinal tract, that is, into the depths of the fasciculi of Burdach. The lesion when at any time it assumes an inflammatory aspect is communicated to the short commissural fibres of the fasciculus, and, step by step, gains in the lumbar region the fasciculus of Goll, which, following the rule, degenerates upwards in its whole length. If things come to pass thus, according to the theory, we should, in a case of recent secondary degeneration of this nature, recognize at the autopsy a lesion limited to the fasciculi of Burdach and not interesting yet the fasciculi of Goll. The future must show if it is really so.

LECTURE NINE.

CEREBRAL, OR SPINAL SECONDARY DEGENERATIONS, IN POINT OF VIEW OF THE LAW OF WALLER: EXPERIMENTS OF SCHIEFFENDECKER, FRANCK, AND PITRES.

Summary:—Wallerean Law of Secondary Degeneration of Nerves.—It is always in a direction from the Trophic Centres.—Examples in Mixed Spinal Nerves; also of their Anterior and Posterior Roots.—Trophic Centres of Motor Nerves are in the Motor Cells of the Anterior Cornua; of Sensory Nerves, in the Intervertebral Ganglia.—In the Spinal Cord the Fasciculi which Degenerate downward are: 1. The Pyramidal Fasciculi whose Trophic Centres are in the Rolandic Region of the Cortex Cerebri. 2. The short Fibres of the Lateral Fasciculi whose Trophic Centres are in the Cord.—The Fasciculi which Degenerate Upward are: 1. The Direct Cerebellar Fasciculi whose Trophic Centres are in the Cord. 2. The Fasciculi of Goll whose Trophic Centres are in the lower part of Cord. 3. The Fasciculi of Burdach whose Trophic Centres are in the Cord.—Degenerations in the Cord by Experiment follow the same law.—Researches of Vulpian, Schieffendecker, Franck, Pitres and others on the Brain and Spinal Cord of Dogs.

GENTLEMEN:

The study of secondary degenerations to which we wish to-day to give the finishing touch, has thus far been almost entirely descriptive. All along we have been demonstrating and describing, but in general we have explained nothing. If we know, for example, that the degeneration of certain fasciculi of the cord is constantly seen in an ascending direction, while that of others is always downwards, we are absolutely ignorant why it is so, and on what principle the law is based. Shall we be content with notions in a manner purely empirical? Or rather, on the contrary, may we attempt the edification of a pathogenetic theory of secondary degenerations? This is what we now propose to attempt.

I. All authorities agree, at this time, with Bouchard, that the *paradigm* of secondary spinal degenerations must be sought in

the domain of the peripheric nerves. This notion is derived from the series of facts discovered from 1849 to 1858, by the English physiologist Waller, and on which rests what is called the Wallerean law. I shall be brief on this subject, which appertains, however, as well to pathology as to physiology, because I shall have occasion to return to it hereafter.

Besides, you will find in the lectures of Claude Bernard on the physiology of the nervous system, every thing of interest about this question, and to which I shall be content to recall for a moment your recollections.

The theory of Waller is founded on the general fact, that when a nerve is divided so as to separate it from its centre, it under goes a change in superior animals, in a certain direction. Thus when a mixed spinal nerve is cut below the confluence of the two roots, it is the peripheric end always which degenerates in its whole extent; and the degeneration takes place in both the centripetal and the centrifugal fibres which compose it. The cause of the sustentation of vitality in the nervous fibres, or, in other words, their trophic centres, must therefore be sought in the direction of the spinal axis. But where are they seated? To discover them it is necessary to make methodic sections as well on the anterior as the posterior roots in various points of their tracts.

1. The section of the anterior roots, at any point, is always followed by a degeneration of the peripheric end, the central portion remaining indemnified; therefore the trophic centre of the roots, and nervous tubes which emanate from them, is in the spinal cord—"probably in the gray substance," says Waller. To-day, thanks to the recent labors of the anatomico-pathologists, we can say with much greater certainty, that it is in the anterior horns, and more definitely still, in their motor cells.

2d. Is it the same for the posterior roots? Certainly not; and this is the most original and unexpected part of the discovery of Waller. The section of the posterior root being practiced, the part which degenerates is not the peripheric portion of the root, that is, the part appertaining to the ganglion, but the central part,—that which is connected with the cord; and the degener-

ation can be followed in the whole extent of the intra-spinal tract of the radicular fasciculi (Burdach's), as far as the gray substance; which proves that the trophic centre for the sensory nerves should be sought in the intervertebral ganglion; and this is so true, that if the ganglion be extirpated a degeneration attacks the entire root, and also all the centripetal fibres of mixed nerves.

You readily comprehend, gentlemen, without making it necessary for me to give further explanation, the application which can be made of these data to secondary spinal degenerations. The fibres of the fasciculi which degenerate downwards below the point of lesion are comparable to the centrifugal nerves that issue from the anterior roots. Of this number are: 1, the *pyramidal fasciculi*, composed of fibres, the trophic centres of which should be in the pyramidal cells of the gray cortex of the region of Rolando; 2, the *short fibres of the lateral fasciculi*, the fibres of origin of which overlap each other from above downwards in the diverse points of spinal gray centre.

On the contrary, the fasciculi which degenerate upwards are similar to the postetior roots whose trophic centre is peripheric. Of this number are: 1, the *direct cerebellar fasciculi*, whose trophic centre is in the cord itself, while the terminal centre is in the cerebellum; 2, the *fasciculi of Goll*, whose trophic centres occupy the gray substance of the inferior regions of the cord, and whose centres of termination are in the gray substance of the bulbar region. Finally, the same interpretation is applicable to the short commissural fibres of the *fasciculi of Burdach*, which equally degenerate from below upwards.

II. The assimilation that we have just made, of the experiments of Waller to the facts of pathological anatomy relative to secondary degenerations, as plausible as it may be able to appear, would be more legitimate still, if the degenerations of the nervous fasciculi, could, with certainty, be reproduced experimentally, as is seen, when nerves are divided.

Now, gentlemen, recent researches tend to establish that this is really the case. The fasciculated secondary degenerations, whether from cerebral or spinal cause, may be reproduced experimentally in animals. To begin, let us consider secondary degen-

erations of spinal origin: the first attempts made by Vulpian and Westphal were not decisive in their results. That was owing to circumstances, as you will soon see, that could not at first be foreseen. The spinal cord of a dog, the subject of the greater part of these experiments, though formed on the model of that in man, differs from it, nevertheless, in some anatomical peculiarities of which I shall show you the principal points, in explaining the researches of the last author, who has written on secondary degenerations, following experiments.

I refer to the labors of Schieffendecker, published in Virchow's Archives in 1876. The author had at his disposal hundreds of dogs, which had served for the experiments of Goltz and Frensburg. Here was material entirely exceptional, not only by reason of the number, but also of the quality of the subjects. Indeed, these animals, in which the cord had been cut transversely, at the level of the twelfth dorsal vertebra, contrary to what is commonly the case in similar mutilations, were, by extraordinary care, kept alive for several weeks and even for several months. Examinations were made after death of hardened pieces, which furnished the means of successful studies of ascending and descending degenerations. In a few words, the following are the results obtained.

In the first place degeneration is a constant fact, as absolutely as in the case of divided spinal nerves. The appearance of the first traces of the morbid process could be fixed, as to date, with the greatest exactness. In the first week already the traces of it are often recognized, but after two weeks the results are absolutely certain. At the end of four or five weeks the highest degree of alteration is attained. I remark on this point, that in man it is only after one or two months that the first traces of secondary degeneration may be recognized; and we must consider, gentlemen, that on this account the observations are much less numerous and less multiplied, than they were in the animals on which Schiefferdecker experimented.

So far as the distribution of the degenerations is concerned, both ascending and descending, they are, according to this author, about the same as in man; there are, however, in the

dog, some peculiarities which I will state to you in two words: 1. In an ascending degeneration the lesions recall exactly what is seen in man under the same circumstances, that is to say, ascending degeneration of the fasciculi of Goll and of the postero-lateral part of the lateral fasciculi, which corresponds to the *direct cerebellar fasciculi.*

2. But it is especially descending degenerations, when compared with those observed in man, that present some peculiarities.

It may be said, in fact, that the pyramidal fibres do not form generally as in man, compact fasciculi (the direct pyramidal fasciculi are however an exception), and in the rest of the antero-

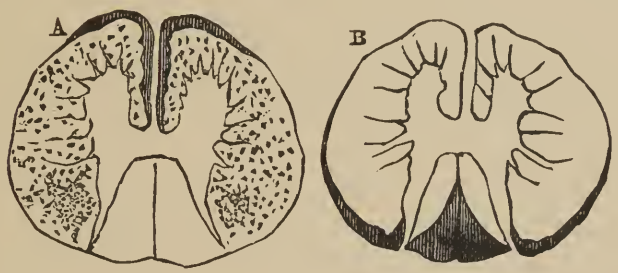

FIG. 30.—A, section of the cord below the lesion. B, section of the cord above the lesion. In A we see the diffusion of the descending sclerosis in the antero-lateral column, except in the anterior part, where the degeneration is well circumscribed in the fasciculi of Türck. In B degeneration of the columns of Goll and of the direct cerebellar fasciculi.

lateral columns the fibres liable to degeneration are somewhat disseminated. Nevertheless, they reunite, in a way, to form a group, more compact, in the pyramidal region of the lateral column, and at this point the fibres degenerate in a long tract, while everywhere else they degenerate only in a short tract.

In sum, *mutatis mutandis*, these secondary degenerations of spinal cause, whether descending or ascending, are absolutely comparable to those seen in man.

III. What has just been said of secondary degenerations of spinal origin, we are able to repeat in respect to secondary degenerations of cerebral origin, when artificially produced in a dog.

It is necessary, in the first place, in regard to the dog, to recall what we have already said of the motor cells in this animal. What are the parts homologous with the regions of Rolando in man? That point, gentlemen, comparative anatomy, thus far, does not seem definitely to have determined. Be that as it may, experimental research on the surface of a dog's brain shows that there are excitable, and motor regions, which, in respect to function, correspond to the Rolandic region in man.

These motor regions in the dog are in a manner grouped about the crucial furrow, and occupy, for the most part, a convolution known as the sigmoid gyrus.

Thus far, you understand, the lesions practiced with the view to determine secondary degenerations in a dog, have related only to the cortex and contiguous parts of the mantle. But it is entirely reasonable to suppose that the results would be similar if the lesions interested the corresponding regions of the internal capsule.

In brief, when the sigmoid gyrus is removed in one hemisphere, secondary degenerations are produced in the dog absolutely in the same way as is seen in man when the lesion has destroyed a certain part of the motor regions.

In this regard it will be sufficient for me to cite some experimental observations: 1. An observation of Gudden, where a lesion of the internal capsule in a young dog was accompanied by a degeneration in the pyramid and in the spinal marrow; 2. An observation of Vulpian who saw, five months after the ablation of the sigmoid gyrus, an atrophy of the corresponding pyramid, with a secondary degeneration; 3, and last, the observations of Frank and Pitres,[1] who give results absolutely identical.

Moreover it is not uncommonly rare to see certain lesions spontaneously developed in that same sigmoid region in dogs, and several times in such cases the existence of secondary degenerations were observed.

You will also find facts of this character in the recent thesis of Issartier, which were developed by Dejerine, Carville and Duret.

Franck and Pitres, to whom we are so much indebted for their

1 See Inaugural Thesis of Issartier, 1878.

important studies on these interesting questions, have gone still further. They devoted themselves to the experimental study of the progress of the morbid process. We now know as a scientific certainty that below the sigmoid gyrus, the excitable part of the cortex, there exists in the mantle a triangular tongue of white substance which unites the gray matter of the cortex to the internal capsule, and which, the same as the region of the cortex from which it springs, responds to experimental excitation, while all the other parts of the mantle are devoid of this property.

Now, gentlemen, according to the experiments of Albertoni and Michieli, this white fasciculus loses its excitability four days after the ablation of the sigmoid gyrus. Thus, separated from its trophic cortical centre, the fibres lose their physiological properties, just as occurs in a peripheric nerve. Pitres and Franck have confirmed these results, Before even the lesion may be appreciable, it certainly exists, as is shown in the loss of function of the nervous elements.

Consequently, you must see that all things concur admirably to support the theory that I have proposed for your assent. There exists, however, it must be admitted, a dark point in our horizon; I mean to say, a notable exception which, thus far at least, seems to contradict the law. I refer to multiple sclerosis where the lesions, even when they are quite extensive, do not produce secondary degenerations. I have proposed meanwhile the hypothesis that this is owing to the persistence still of the axes cylinders in the foci of multilocular sclerosis. But I do not guarantee the absolute value of my explanation, though it rests indeed on a real observation. Possibly, for want of sufficient examination, certain descending degenerations may not have been seen. Whatever may be the hypothesis, I believe that it will be necessary to revise, on this account, the pathological anatomy of multiple sclerosis. However, that is a matter which I barely refer to now. We shall have occasion, certainly, to refer to this subject again.

LECTURE TEN.

DETERMINATION OF THE TRACTS OF THE WHITE FASCICULI OF THE SPINAL CORD BY THE STUDY OF SECONDARY DEGENERATIONS.—EXPERIMENTAL ANALYSIS OF FUNCTIONS OF THE PYRAMIDAL FASCICULUS.

Summary:—Clinical Aspect of Secondary Degenerations.—Structural Anatomy of the Spinal Cord.—Schema of Bouchard.—White Fasciculi are all subject to Degeneration.—Physiological Experiments unsatisfactory.—Relation of Function of Fibres in Antero-Lateral Columns to Centrifugal Nerves.—Influence of the Brain (will) on Excitability of the Cord.—Voluntary Excitation transmitted by the Pyramidal Fasciculi.—Experiments to prove this by Vulpian and Woroschiloff.—Explanatory Plates of Sections of the Cord to show the Functions of its Constituent Parts.

GENTLEMEN:

You have not forgotten, I am sure, the proposition that I laid down at the beginning, even, of our studies of secondary degenerations. These lesions, I said to you, are interesting not alone in point of view of pure pathological anatomy; they are, during life, the occasion of particular functional troubles, which are superadded to the symptomatology of the primitive lesions from which they originate, and sometimes even dominate it, and on this account they merit the special attention of clinicians. The time is now come to justify our assertion, and to show you the practical side of the delicate and complicated studies that we have thus far pursued. It is therefore the clinical aspect of secondary degenerations that we now proceed to consider. But, beforehand, there are yet two points on which I wish, by way of preparation, to fix your attention.

In order to comprehend the nature and origin of the functional troubles which are attached to descending degenerative lesions of the various fasciculi of the spinal cord, we must certainly invoke,

especially, the anatomico-pathological notions that we have laboriously collected on all points relating to these lesions.

But to bring to a successful termination our undertaking, that would not yet suffice.

1. It is necessary, in fact, to be established on some points relative to the structure of the spinal cord, and to the arrangement of the nervous fibres and cellular elements which compose it, and their mutual relations. It is true that as we have passed along we have presented certain documents which concern, very especially, this subject, but it is important now, I think, to consider the question no longer in part, but as a whole.

2. On the other hand, we must appeal to the notions furnished by experiment on the physiology of the spinal fasciculi. It is true that the anatomico-clinical methods are only appealed to, to sustain, as a last resort, the physiology of the diverse parts of the nervous system in what relates to man; it is equally true that experiments on animals furnish in this regard important major data by showing the way in which anatomico-clinical investigations should be directed.

I. I will commence then, at once, with the first point. It has often been said that morbid anatomy illuminates normal anatomy, and that it can decide even the questions that the latter by its own showing would not be capable of resolving.

The anatomico-pathological history of secondary degenerations justifies, in part, that proposition. In what concerns the arrangement of the constituents of the spinal cord it furnishes, in fact, certain information which yields in no respect to the data of pure anatomy and sometimes even greatly surpasses it.

These then, in a few words, are some views which I wish you to understand concerning the structural anatomy of the spinal cord.

II. From what has been said, you can readily conclude that the white fasciculi, which compose the external portion of the cord, are all subject to secondary degeneration when their constituent fibres are interrupted in their tracts. But in regard to this there are two categories to establish. A. Some of these fasciculi are constructed of the long fibres which extend through the entire

length of the nerve-axis. These are the seat of secondary degenerations of *long tract.* Of this number are: (*a*) the pyramidal fasciculi (descending degenerations); (*b*) the fasciculi of Goll (ascending degeneration); (*c*) the direct cerebellar fasciculi (ascending degeneration).

B. The other fasciculi are constructed of short fibres. Of this number are: (*a*) the antero lateral fasciculi, or, better still, what remains of the fasciculi when an abstraction is made of the pyramidal fasciculi, and the direct cerebellar fasciculi; they degenerate downwards; (*b*) the fibres proper of the fasciculi of

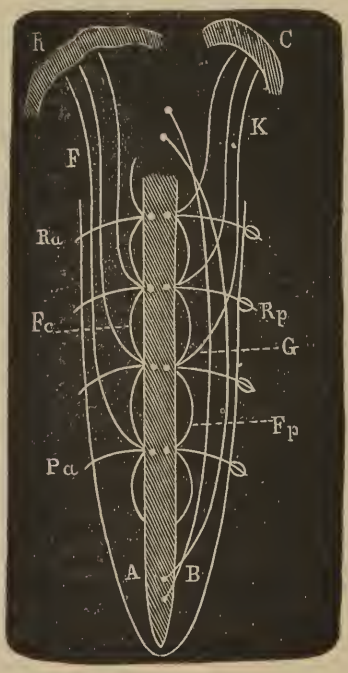

FIG. 31.—A., anterior fasciculi. B., posterior fasciculi. R., convolutions of Rolando. C., cerebellum. R.a., anterior roots. R.p., posterior roots. F., pyramidal fasciculi. F.c., intrinsic centrifugal fibres (anterior columns). K., extrinsic centrifugal fibres (direct cerebellar fasciculi). F.p., intrinsic centrifugal fibres (fasciculus of Burdach). G., long posterior fibres (columns of Goll).

Burdach, which degenerate upwards. In these two instances the degeneration is of *short tract*.

If now, possessed of these data, we consider the construction of the spinal cord in its entirety, we can form a *schema* which shall not differ notably from that which Bouchard has presented in that work of his to which I have already several times referred.

The spinal cord may be considered as constructed essentially of a gray axis around which are grouped all the other parts.

The fundamental parts of this axis, that is to say, the ganglionic cellular elements are motor or kinesodic, and sensitive or œsthesodic ; the latter to transmit impressions.

Finally, these two elementary structures are bound together in a thousand ways by the intermediation, it is thought, of a nervous plexus. However that may be, the gray axis gives origin in front to the anterior roots, which are in direct connection with the cells of the anterior cornua by means of the prolongation of Deiters. On the other (rear) side the posterior roots are in relation with the œsthesodic cells.

But the cord, thus so very complicated in its structure, is still quite incomplete. It is important to indicate the relation of the gray axis with the diverse fasciculi of white fibres which compose its medullary portion. I must now consider, successively, the antero-lateral and the posterior fasciculi. I will begin with the latter.

Among the fasciculi which compose the posterior columns some are called intrinsic, others extrinsic, according to the nomenclature proposed by Bouchard. The intrinsic are displayed in two groups: 1. In the fasciculi of Burdach the intrinsic fibres are the short commissures which place in communication the œsthesodic cells in the whole length of the cord. Each of the fibres has its trophic centre below and degenerates, as you know, upwards. 2. In the fasciculi of Goll, on the contrary, we find the long commissural fibres which degenerate also from below upwards and whose trophic centre is necessarily situated lower than the centre of termination. These intrinsic fibres of the posterior spinal system have with the encephalon, only indirect relations and as yet but little known.

So far as the extrinsic elements are concerned they are represented by the direct cerebellar fasciculi, the inferior extremities of which plunge into the gray substance where they have their trophic centres, while above they terminate in the cerebellum which is thus put in a direct relation with the spinal cord proper.

In the antero-lateral fasciculi we must also consider the intrinsic and extrinsic parts. The first are represented by the short commissural fibres which, probably, connect the motor cells of the various stages. The trophic centre of these fibres is above their centre of termination, since they degenerate from above downwards.

The extrinsic fibres of these fasciculi are simply both the direct, and the crossed pyramidal tracts, and in regard to their direction, origin and termination, the anatomico-pathological and experimental study has been the most satisfactory. It appears no longer doubtful that these fasciculi serve as a bridge—as a veritable commissure, establishing direct connections between certain regions of the brain proper and the kinesodic cells of the diverse stages of the gray axis. The origin of the fibres which constitute these fasciculi is in the cortex of the cerebral convolutions, probably in the pyramidal cells of the gray substance. These fibres traverse the encephalon, the protuberance, the bulb and finally the entire length of the cord, bearing no relation to other tracts than that of contiguity, and in this way they disappear successively in their descending course through the various regions of the cord, entering into connection with the motor cells, by the intermediation of an anatomical arrangement not yet fully understood.

III. Let this suffice for anatomy. I will next consider the physiological side and show the principal results obtained by experiments in regard to the functions of the medullary fasciculi. I will commence with the antero-lateral columns.

The idea would be that physiology could enlighten us on the particular functions of each of the secondary fasciculi which the anatomy of development and pathological anatomy have shown us to be segregate in this part of the spinal medullary mantle. Unhappily experiment has not made account, generally, of these

distinctions. I shall have, nevertheless, occasion to speak of some attempts recently made in this regard, and which exhibit the part played by the pyramidal fasciculi in the transmission of voluntary incitations.

1. In respect to the degenerations which take place in the antero-lateral fasciculi, when their fibres are interrupted in their tract, we have already seen that the fibres which compose them follow the same laws as the peripheric centrifugal nerves. Now, may these antero-lateral fasciculi show a closer relation still to the motor nerves in other qualities? Not absolutely, for we know that the section of centrifugal nerves is followed by muscular atrophy, while the interruption of function in the antero-lateral column is not followed by a like result. This proves that the anterior roots are not simply prolongations of the nerve tubes of these fasciculi; they are separated from each other by the ganglionary cells, which are their veritable trophic centres. There is then no identity and we need not count on more than mere analogies.

Are the antero-lateral fasciculi excitable like the centrifugal nerves? In other terms, is the irritation of their nervous fibres by mechanical, chemical or electrical agents followed, as is the case when the nerves are thus treated, by muscular contractures? It is now affirmatively established, by the researches of Vulpian and the more recent ones of Fick and Engleken that the antero-lateral fasciculi are excitable like the centrifugal nerves; but the effects of this excitation are much less accentuated in regard to the generalization and intensity of the contractions. It is remarkable also, that the physiological, voluntary, excitation limits very much, in such a case, artificial excitation. The cells of the anterior cornua are, doubtless, an obstacle which restrains the propagation of excitations unto the anterior roots.

But it was, especially, interesting to ascertain if the pyramidal columns are *more*, or *less* excitable than the anterior fasciculi. Now experimentation in these researches encounters very serious difficulties. The authors agree to recognize that in the dog the excitability is more pronounced in the anterior fasciculi. It is possible that it may be so in the dog, where, according to the

observations of Schieffendecker, the fasciculi of Türck which appertain to the system of the pyramidal fasciculi and composed, consequently, of long fibres, would have in this regard considerable importance.

However, is it the same in man where, on the contrary, the fasciculi of Türck are of small import, the anterior fasciculi proper being composed of short commissural fibres, that is, forming a fasciculus the tract of which is interrupted every moment by the ganglionary cells? It may be doubted that it is so. Besides, we know the excitability of the prolongations of the pyramidal fasciculus in the mantle of the cerebral cortex, and we know also that it constitutes there a nervous tongue, subjacent to the Rolandic convolutions, endowed with very manifest excitability; we may then admit that in man that same excitability must exist in the whole extent of the prolongation of these fasciculi in the spinal cord.

2. It is generally accepted at this time in experimental physiology, that the transmission of voluntary motor excitation is made exclusively through the white antero-lateral fasciculi. This is at least what the experiments of Vulpian, contrary on this point to those of Schiff, seem to have established in a peremptory manner.

In the frog (and *a fortiori* in mammifera) the section of the lateral fasciculi in the dorsal region (the posterior fasciculi and the gray substance being spared), abolishes, in a definite manner, the voluntary movements in the posterior extremities; that is to say, not only on the day of the experiment, but subsequently. On the contrary, the section of the posterior fasciculi and the gray substance does not prevent the persistence of voluntary movements in hind feet.

B. It is also very important to consider the result of spinal hemisections. The experiments of Schiff, repeated by Vulpian and others, have modified in this regard the old doctrines which have existed from the time of Galen. It was believed that transmission was absolutely direct. Now we know that they are both direct and crossed. When the lateral moiety of the cord is cut in a guinea-pig, for example, the paralysis is at first very pronounced

on the corresponding side, and there exists a slight paresis on the other side. But very soon, if the animal survives, the paralysis is diminished on the side of the hemisection, though it persists to a certain degree. This is due to the existence of commissures which unite the antero-lateral fasciculi of one side to the corresponding ones on the other side. But if you practice a second hemisection, on the side at first respected, a paraplegia becomes complete in both members. It is then, necessarily, by the antero-lateral columns alone that voluntary motor incitations are conducted.

It is not therefore, as it is in regard to sensibility, a matter of indifference by what elements of the cord the conduction is effected.

Thus, while the transmission of sensitive impressions is yet possible, after the cord has been subjected at different levels to two hemisections in an inverse manner, there exists at the same time a complete, absolute paraplegia of motility.

IV. But do the antero-lateral fasciculi as a whole transmit the mandates of the will, or is that function performed by the pyramidal fasciculi? Experiment has nearly always failed when attempting this analysis. "A vivisection sundering isolately and completely the anterior or the lateral fasciculi," says Vulpian, "is impossible to realize."[1]

It suffices, to be convinced of this, to examine the configuration of a transverse section of the spinal cord. Nevertheless, gentlemen, latterly Woroschiloff, by using the more perfect instruments which are daily employed in the laboratory of Ludwig, has been able to make very varied sections of the cord in a rabbit, both as to the seat and extent of the parts involved, and has in this way been able to produce combinations which have enabled him to isolate the function of the pyramidal fasciculi in the transmission of voluntary incitations, and has confirmed by this means the results obtained heretofore on the same subject by Miescher, Nawrocki and Dittman.[2]

It was the aim in the experiments of Woroschiloff to ascertain the part taken by the various spinal fasciculi in the execution of

1 Diet. Encycl. des sc. Med. Art. Moelle.
2 Bericht d. gesselsch. d. Wissensch. zu Leipsig, 1874.

some voluntary movements of easy analysis, such as jumping, running, walking, etc.

The extent and configuration of the lesions were studied by him with the greatest care in hardened sections and reproduced photographically, in the work in question.

Let me now show you some of the results which these experiments have furnished. They particularly interest us because, as you see, they isolate the special functions of the pyramidal fasciculus.

FIG. 32.

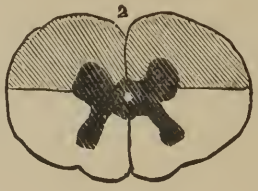

FIG. 33.

1. The section of the posterior fasciculi do not modify the voluntary movements.[1]

2. It is very remarkable that all of the anterior moiety of the cord can be divided without being followed by any modification in the execution of voluntary movements, which shows us that the posterior moiety is sufficient for their transmission. There are then no long fibres *directly cerebral* in the anterior fasciculi.

FIG. 34.

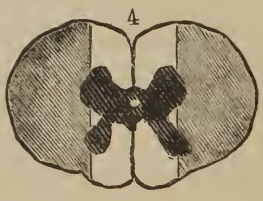

FIG. 35.

3. The gray substance may be cut in its whole extent, the antero-lateral fasciculi remaining intact, without any modification whatever in the voluntary movements.

1 In the schema the shaded parts represent the extent of the medullary section.

4. If, on the contrary, the gray substance is intact and the antero-lateral fibres are divided on both sides, the posterior members are completely paralysed. This shows you that the gray substance is not sufficient for the transmission of voluntary incitations.

FIG. 36.

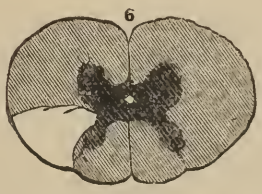

FIG. 37.

5. In the case of a section of the lateral and posterior fasciculi the animal can use only the anterior members. The posterior extremities are completely paralyzed.

6. Finally, if the section is total, except a single lateral fasciculus, the inferior member of the sectioned side is completely paralysed. On the contrary, on the side where the lateral fasciculus is spared the member still obeys the will; if it be extended the animal withdraws it. It is thus demonstrated that the pyramidal fasciculi transmits the voluntary incitations.

But it is possible that the function of the pyramidal fasciculi may not be exclusive; doubtless, however, they represent the easiest and most habitual way of such transmission. Nevertheless, when the tract is severed, it is reasonable to suppose that other tracts may obey, at least in certain cases, the mandates of the brain. This is a point that I shall have to take up again.

V. In order to finish the subject of these physiological prodromes, there remains for me to say only a few words on the influence exercised by the pyramidal fasciculi on spinal reflex activity.

Already our studies relative to development have led us to recognize that the pyramidal fasciculi should be considered as the grand commissure by which functional relations are established between the brain and the spinal cord. The latter exists in away, isolated while the fasciculi are yet undeveloped; and up to that

time the movements of the new-born are entirely of the reflex order. The reflex movements are, on the other hand, established on the second plane when the development of the pyramidal fasciculi is accomplished, and after that the voluntary movements predominate.

The moderating influence of the brain on reflex actions is seen very clearly when observations are made after the section of the cord. But it is easy to show that it is by the antero-lateral fasciculi that such influence is exerted. In fact the section may be so made that sensibility is preserved; moreover one side only of the cord may be affected. The antero-lateral fasciculi are then really the conductors of voluntary incitations.

Nevertheless experimental physiology has not yet decided if it be by the way of the anterior or the pyramidal fasciculi that this influence is exercised. But we shall see that pathological facts resolve the question that physiological experiments leaves undecided.

Such are the preliminary notices that I have felt it my duty to present to you relative to the functional action of the diverse fasciculi of the spinal cord—as they are revealed by experimental researches. We are able now, to consider successfully, the clinic of secondary degenerations.

LECTURE ELEVEN.

GENERAL SEMINOLOGY OF SECONDARY DEGENERATIONS OF THE PYRAMIDAL FASCICULUS.

Summary: — Symptomatology of Secondary Degenerations. — Study of Apoplectic Hemiplegia. — Prognosis. — Focal Lesions. — Anatomical Description of Central Masses of the Brain.—Arteries of the Region: their distribution.—Terminal Arteries.—Miliary Aneurisms.—Hæmorrhage, commonly, arises from their Rupture.— Diffusion of the Blood in Ventricles a cause of Sudden Death.—Effects of Pressure, or Destruction, on the Internal Capsule, and of the Pyramidal Fasciculus.—Tardy Contracture in Hemiplegia the Pathognomonic Sign of Lesion of the Pyramidal Fasciculi.—Tendinous Reflexions.—Provoked Trepidation.—Provoked Spinal Epilepsy.—Foot Clonus.—Prodromic Period of Contracture.—Hand Phenomenon.—Associated Movements.

GENTLEMEN:

I propose to-day to consider secondary degenerations in a clinical point of view. In other words, I will attempt to exhibit a tableau of functional disorders which are attached to these lesions and by which they are revealed. Then after placing under contribution the data of the experimental order, which I briefly brought before you in my last lecture, I will attempt the physiological interpretation of the symptoms.

I. You are doubtless aware that this subject has been treated in a very luminous manner by Professor Bouchard, in that treatise of 1866 which I have so often put under contribution, and the symptomatology of secondary degenerations therein is incontestably one of its most original parts.

On several occasions in this course I have given an exposition of the symptoms connected with secondary degenerations and for the most part, conformably to that which is given by Bouchard. I shall do the same thing at this time; nevertheless, several new data have been introduced since 1866 into this interesting chapter, which I will bring before you. I shall also propose some modifi-

cations of that which relates to the physiological interpretation of the phenomena.

The necessary divisions to establish to aid our demonstrations are all naturally traced. We will pass successively in review consecutive degenerations of cerebral origin, and those which indicate, as a cause, a spinal lesion.

II. To thoroughly comprehend the practical interest of the study we now undertake, it is convenient to consider the state of a patient suddenly stricken with hemiplegia, in consequence of the the formation of a hemorrhagic focus or softening in the brain itself.

To be more precise, let us take a case of intracephalic hemorrhage of average intensity. What we say of it, we shall be able, with some modifications of a secondary character, to apply readily hereafter to a case of apoplectiform softening.

I will suppose also that the patient has surmounted the first difficulties of the situation. The comatose phenomena the intellectual torpor of apoplexy, the fever—the "precocious" contractures, the erythema of the gluteal regions,—when, in fact, all the symptoms of evil augury, so far as they have existed, have actually disappeared; in a word, about the twelfth or fifteenth day after the attack, when it has become certain that, without some fortuitious complication, the patient will live.

But here, gentlemen, a most important question presents itself to the mind of the physician, and about which he will certainly be interrogated by the friends, or even by the patient himself. Now there still exists a complete motor paralysis, absolute, or nearly so, of one side of the body. The superior member is completely flaccid and falls heavily on the bed when it is elevated and dropped. The patient, besides, is unable to make any movement with any part of the arm or hand. It is the same with the corresponding lower limb, though it may be slightly less so. Finally, the face of the same side, in its lower region, is also seriously affected. Thus, the labial commissure of the sound side is elevated while at the same time the lips are slightly separated; the paralysed side is drooping, while the lips are thin. It is unnecessary to enter into further details.

Now the question to resolve is this: the patient will live, he will recover doubtless, for the most part, the activity of his intellectual faculties; but will he ever recover the use of his paralysed muscles? Will he be able to make use of his hand or his arm? Will he be able soon to leave his bed, stand erect, and walk? Or inversely is he henceforth condemned to motor inaction and the loss of the regular use of his members? In a word, must he continue in this infirm state, confined to his chamber and obliged to depend upon the assistance of others to accomplish the most imperious acts of his ordinary life?

Such is the problem which arises in the particular conditions that we have described. Well then, in such a case, the intelligent physician, while at the same time he endeavors to find in the examination of the patient indications sufficient to furnish him a solution of the very difficult question, will not fail, even instinctively, to call to mind the anatomico-pathological and physiological knowledge appropriate to enlighten him. He will think anatomically, if I may so express myself, physiologically, and at the same time clinically. He will not be content with empirical data but for want of better, for he wishes even in a limited way, to penetrate the principles of things. We cannot do better, I think, than to imitate him: let us then conceive the state of the tissues affected in the patient who is thus before us, and about whose future prospects we must give an opinion.

III. This leads us into a digression, but I do not doubt that after following the devious route by which I propose to lead you, the task which we attempt to fulfil will be rendered much easier. It concerns you remember, in the example chosen, an intra-cephalic hæmorrhage of the most common order. Where is the focus seated? What parts has it simply compressed? What parts has it destroyed? Ninety-five times in one hundred it will be found as I now proceed to assume.

Let us recall some of the anatomical peculiarities which exist in the region of the central masses; for it is in this region that the effusion takes place in a great majority of cases.

Let us examine anew the horizontal section that I have several times shown you. I call your attention particularly now to the

relations of the lenticular nucleus. Within it is limited by the internal capsule, without by another white tract, the external capsule, which separates it from the outer wall (*Claustrum*), and from the region of the island of Reil.

Now, gentlemen, on these convenient pieces, already hardened in alcohol, it is easy to show you that the external face of the lenticular nucleus, in its anterior third especially, is but very loosely united to the external capsule. In reality there is no intimate anatomical connection between this thin white band and the external face of the gray nucleus. There exists at this point a sort of virtual ventricle, and if it is in this region, as was remarked long ago by Gendrin, that the sanguine effusion begins to collect in ordinary cases, the reason of it is not difficult to comprehend. To this same region appertain in fact, the nourishing arteries, the lesion of which inaugurates in a manner the intra-cephalic hemorrhage.

If in a brain previously injected, you will study, guided by the important labors of Duret, the arrangement of the nourishing arteries of the cerebral masses you will find in particular what follows:

The most important of the arterioles proceed from the sylvian trunk (middle cerebral artery), from which they are detached, perpendicularly, and penetrate the nervous substance at the level of the anterior "perforated space." We see there a series of small apertures, each one of which gives passage to an arteriole, and carefully note, contrary to what takes place in the cortical arterial vessels destined to nourish the gray substance and the mantle, that the vessels which are insinuated into the thickness of the central masses are not in respect to their structure or calibre, capillaries, but veritable arteries.

The distribution of the lenticulo-striated vessels after they have penetrated the central masses can be easily shown by a very simple dissection. The gray cortex of the convolutions of the island of Reil, the subjacent white substance, the claustrum and the external capsule should be removed, and, in this way, the convex surface of the third segment of the lenticular nucleus is laid bare and on this surface the fanlike expansion of the

lenticular arterioles is clearly seen. Those in front are the most voluminous; all are directed from before backwards and from beneath upwards, penetrating towards the superior extremity of the nucleus, its interior, where they disappear.

In order to understand the ulterior tract of these vessels and their ramifications in the interior of the gray nuclei, it is necessary to make transverse sections through the nuclei. The examination of one of these sections, if well chosen, will suffice for the end we have in view. I refer to a section made slightly in advance of the chiasma. This exhibits one of the largest and most important of the striated arteries, by reason of its fundamental character in the cerebral hemisphere. After having penetrated the depths of the third segment, it traverses the superior part of the internal capsule, and reaches as far as the caudate nucleus. The other arterioles are arranged nearly after the same plan.

These are the *terminal arteries* that is, those that do not communicate with each other, nor with the cortical arteries. An injection made with too much force easily ruptures them and produces small foci which are similar to those seen in cerebral hæmorrhage.

Finally, they are, particularly, subject to a form of arteriosis which leads to the formation of miliary aneurisms—that preparatory lesion of intracephalic hæmorrhage. It is very common in those who have previously been stricken by a cerebral hæmorrhage, to extract from the apertures of the "perforated space," a certain number of arterioles bearing miliary aneurisms.

When a hæmorrhage occurs owing to the degeneration of these arteries, which is very frequently the case, the blood is diffused in the virtual ventricle, of which I have spoken, between the lenticular nucleus and the external capsule. In this way are formed flattened foci, which when the effused blood is reabsorbed, are seen in the form of an ochreous, linear cicatrice limiting the external contour of the lenticular nucleus.

When the focus in question is still recent, provided the effusion has been notable, the gray nucleus is necessarily pressed towards the ventricle on account of the greater resistance that the cranial walls offer on the side of the insula (island of Reil). You see

then, that if matters stop there, no important point will be destroyed; the internal capsule, in particular, will only be compressed mediately. In such a case, notwithstanding the intensity of the first symptoms, if the patient survives the shock it will be remediable, that is, the integrity of movements will sooner or later be quite restored.

But, on the other hand, the focus may be enlarged, and following the direction of the arterioles, cut across the internal capsule and penetrate the ventricular cavity. In such a case the condition is most grave; the patient, generally, succumbs, and by this fact the question of the extent of the injury is decided.

If the ventricular inundation does not take place, if the fibres of the capsule have been torn only, provided that the rupture apertains to the pyramidal fasciculus, the case is still very serious,— not in regard to the life of the patient, which is unmenaced, but in what concerns the return of voluntary movements in the members stricken with hemiplegia. Very certainly in that event the integrity of movements is definitely compromised.

Such a destructive lesion is followed, necessarily, by the development of a descending spinal lesion which, in its turn as I will proceed to show, is the fatal cause of the indefinite persistence, more or less complete, of the motor incompetency of the paralyzed members.

Thus you see, that a few millimetres more or less, in the extent of the lesion following a certain direction, is not an indifferent matter. In proportion as the fibres of the pyramidal fasciculus are spared, whatever may be the extent of the focus, the lesion is reparable. It is not so or but little so when the fibres of the fasciculus have not only been compressed but destroyed in any point of their tract. Such is a summary of the situation.

IV. It is a sufficient explanation also of the pathological anatomy. The question presents itself now as follows: Is it possible to recognize clinically, with exactness, in persons stricken with hemiplegia, for say a fortnight, that the pyramidal fasciculus has been destroyed in any part of its range; or, in other words, is secondary degeneration revealed by characteristic symptoms? We can reply in the affirmative. It is certain that the existence of

secondary degenerations, in the conditions which we have described, may be readily recognized. The great pathognomonic symptom in such a case is that unity of the phenomena which is commonly designated as the *tardy contracture of hemiplegia*. It is necessary then to give our whole attention to this remarkable symptom. But it does not usually manifest itself in a decisive manner until about the second or third month from the attack. Must we wait, to give an opinion, until the period is complete? Do there not exist, in the symptomatology of apoplectic hemiplegia, indications less tardy and capable of revealing the existence of consecutive spinal lesions?

In fact, gentlemen, the period of tardy contracture in incurable hemiplegia, is preceded by a prodromic period, during which certain phenomena are witnessed, which, if they do not permit us to assume the certainty of a degeneration, at least render its presence very reasonable. But the appearance of these phenomena should be provoked by the observer. They are signs which we must learn how to make apparent; they are only revealed by certain procedures. To-day I shall limit myself to reminding you of one of them, the oldest introduced into the semiology of neuropathology, and which is known in France as provoked trepidations, provoked spinal epilepsy. German authors, call it the foot phenomenon (Fuss-phœnomen), or the foot *clonus*.

But this sign appertains to the French clinic. From 1863, as is shown by the recorded ohservations of that period, it was employed daily in the service of Salpétriére, by Vulpian, myself, and by our students. From that time we have not ceased to study that phenomenon in its relations with the different affections of the nervous centres, and have sought its signification. Thus, I have for a long time shown that it is constantly absent in the spinal paralysis of infancy, and also in the motor incapacity connected with ataxic tabes, and in other similar affections; while it is present in cerebral or spinal paralysis, in which contracture exists, or where it is imminent.[1]

This phenomenon is produced on a patient, by lifting the paralysed lower limb and placing the left hand in the ham in

1 Dubois, Thése de Paris, 1868.

such a way that the leg swings free, and then roughly lift the point of the foot with the other hand; immediately there arises a series of agitations which together constitute a sort of rythmical movement,—of oscillating trembling, more or less regular or persistent.

Spinal trepidation is of so much greater interest in the fact that not a trace of it exists in the normal state. Thus, Berger,[1] in 14,000 healthy persons, mostly soldiers, saw the phenomenon but three times. I will repeat also, for a purpose, that in the pathological domain it is not a common sight, since in some spinal affections it is absent, while in others, as a rule, it is present. It is, in short, one of the characters of the group of spasmodic paralyses; and central hemiplegia, with secondary degeneration of the pyramidal fasciculi, belongs to this category.

When tardy contracture exists it is nearly constant. But several weeks often precede it. In a patient now confined in the infirmary of Salpétriére it began to manifest itself eight days after the attack; and fifteen days later the rigidity of the inferior member inaugurated, gradually, the series of spasmodic accidents. In another patient they only began a month after the attack, and muscular rigidity began to be seen in the course of the second month. Dejerine has recently announced that this symptom is revealed occasionally in both of the inferior members, and we shall see that it is the same sometimes in contracture. Among hemiplegics who still enjoy some movements, this same trepidation, which involves in certain cases the entire limb, may also be seen on the occasion of voluntary movements.

This is a reflex phenomenon, which I propose also to demonstrate more at length hereafter. Let it suffice for the moment to remark, that its intensity is provoked by the employment of strychnia, lessened, on the contrary, according to Berger, by that of opium.

An analogous phenomenon is sometimes produced when the hand of a hemiplegic is quickly raised by the ends of the fingers. Often also these patients, on lifting the paralysed arm, feel a similar trepidation, like that which is excited in the inferior

1 Arch. d. Heilk., 1879, No. 4.

member under the same circumstances. But the *hand phenomenon*, provoked or spontaneous, is much more rare than the corresponding one, known as the *foot phenomenon*.

These two signs, as we shall show you, appertain to the same category as those which have been recently introduced into the semiotics of spinal affections by Westphal, and later by Erb, under the collective designation of *tendinous reflexions*.

LECTURE TWELVE.

TENDINOUS REFLEXIONS IN SECONDARY DEGENERATIONS OF CEREBRAL ORIGIN. INFLUENCE OF NUX VOMICA ON THE PRODUCTION OF CONTRACTURE.

Summary:—Spinal Trepidation.—Foot Phenomenon.—Tardy Contracture.—Tendinous Reflexions.—Patella Reflexions.—Knee Phenomenon is normal, very marked in the New-born.—Knee Symptoms abound in Locomotor Ataxia and in Anterior Polyomyelitis: is exaggerated in Spasmodic Paralysis, Suppressed by Counter Irritation.—Tendon Sign is a Spinal Reflexion: its Centre is in the Cord between the Fifth and Sixth Lumbar Nerves.—Tendon Reflexions in Superior Extremities rarely Developed in the Normal State.—Indications furnished by Graphic Demonstrations.—Prodromic Period of Contracture.—Associated Movements.—Syncineses.—Effects of Strychnia in Paralyzed Members.—Researches of Fouquier with Nux Vomica.

GENTLEMEN:

I said to you when I finished my last lecture, that in permanent hemiplegia of cerebral orign, the period of tardy contracture is preceded by a prodromic period, during which certain phenomena are manifested which, if they do not always permit us to affirm the existence of degeneration, render it nevertheless highly probable.

These phenomena, I added, are not spontaneous; their appearance must be provoked by the clinician. They are, in other terms, signs which must be made evident by certain very simple procedures. Beyond the case that we now consider, we shall, in the course of our studies on cerebral and spinal organic affections, meet again very often these same phenomena with all the characters that I seek to place in relief, though, indeed, nearly always under conditions, relatively, much more complex. On this account it is necessary to avail ourselves of the occasion which now offers, for the examination of these phenomena in the most favorable circumstances for physiological analysis.

I have already spoken to you of one of the oldest of them,

introduced into neuro-pathological semiotics. It is now, you are aware, designated in France as *provoked trepidation*, *provoked spinal epilepsy*, and in Germany as the *foot phenomenon*, *clonus of the foot*. In the pathological domain it is not by any means an ordinary phenomenon. As a rule it is absolutely absent, or very attenuated, in certain spinal affections as, for example, locomotor ataxia, acute or chronic anterior poliomyelitis (infantile paralysis, protopathic spinal progressive muscular atrophies, and all other affections of the same category), while in others it is habitually present. It is, in a word, one of the characters of the clinical group in spasmodic paralyses; and, cerebral hemiplegia with degeneration of the pyramidal fasciculi belongs to that.

When once the tardy contracture is established the spinal trepidation is, save in exceptional circumstances, a constant symptom, and even precedes it by several weeks.

Thus in a patient now in Salpétriére, and stricken several months since with hemiplegia, a provoked trepidation was manifested eight days after the attack, and it was only fifteen days later, that is at the end of about three weeks, that contracture began to appear.

In another patient the foot phenomenon did not show itself until a month after the attack, and contracture was not established until near the end of the second month.

Moreover, Dejerine, as I have already told you, has quite recently made the very interesting and very exact remark, that the inferior member of the non-paralysed side becomes sometimes the seat of provoked trepidation. It is not rare in such a case to see a permanent contracture developed in this member, in such a manner, that the hemiplegia becomes complicated with a paraplegia with rigidity, which places an obstacle nearly invincible in the way of station and walking, and the patient is then confined to his bed to the end of his days.

Finally, in hemiplegics who yet are capable of some movements, this same trepidation of the foot, and which at times extends to all parts of the member, may manifest itself on the occasion of a voluntary movement.

Provoked or spontaneous, the trepidation in question, as I shall demonstrate, is a phenomenon of reflex order. Let it suffice for

the moment to remark that its intensity is increased under the influence of strychnia; that it becomes attenuated on the contrary by the employment, in full doses, of bromide of potassium, and opium also, according to Berger; a statement which, it seems to me, is much less established than the other.

II. The *clonus* of the foot and that of the hand appertain, as it will be easy for me to show you, to the same category as those we proceed to consider, and which have recently been introduced into the semiotics of cerebro-spinal affections, by Westphal first and afterward by Erb.[1]

These new phenomena are collectively designated as *tendinous reflexions*. Of all of them, the best studied, the easiest to provoke, and which at the same time is practically the most interesting, is known as the *patella reflex*, *reflex of the rotulian tendon*, *knee phenomenon*, *clonus of the knee*, etc. I will explain the method of exciting it. The limb to be tested is upheld as in the former method, by placing the left hand in the ham; the leg hanging free, the tendon is hit about its middle by the cubital border of the right hand, or, better still, by Skoda's percussion hammer. A simple fillip with the middle finger at the point indicated will produce the same result, at least in certain pathological circumstances. Almost immediately after the shock, the leg of the patient is elevated more or less brusqely—describing a curve greater or less as the case may be, and then subsides to its dependent position. Sometimes, though, a single shock is followed by two or three successive oscillations; the phenomenon has then acquired its highest degree of intensity.

It is important to note that contrary to what takes place in provoked trepidation of the foot, the knee phenomenon belongs to the normal state. In every sound person, with rare exceptions, it exists to a certain extent; thus in the statistics of Berger, of whom I spoke in my last lecture,[2] the rotulian reflex did not fail one *per cent.* Eulenberg[3] has made the interesting remark that it is very accentuated in the new-born on the first day, and that it becomes less so, generally, at the end of a few weeks.

1 Arch. f. Psych., vol. v., 1875.
2 Centralblatt f. d. Nervenkr. 1879.
3 Ibid. 1878.

This last fact, if it be confirmed, has an important bearing, since it seems, uniquely, to show that at this period the phenomenon is a spinal reflexion. We have not forgotten, indeed, that in the new-born the pyramidal fasciculi are not yet completely developed, and consequently the moderating influence exercised by the brain on spinal reflex actions cannot yet be exerted.

However that may be, it follows from what is said above, that the knee phenomenon does not acquire a pathological signification except under the following circumstances: 1, where it is completely at fault as is commonly the case, for example, in locomotor ataxia, or in anterior poliomyelitis; 2, or it is notably exaggerated as in spasmodic paralyses. In hemiplegics menaced with contracture, it precedes even quite often, the appearance of the foot phenomenon; and if it happen to be a little too pronounced it is not without significance, considering the still less intensity of the same symptom on the non-paralysed side.

III. As its name indicates the knee phenomenon is certainly of the reflex order; it is a spinal reflex, and already some clinical observations allow us to foresee it. Thus Erb has remarked,[1] that on percussing the rotulian tendon so as to provoke the knee phenomenon, there sometimes occurs, simultaneously, a movement of adduction in the opposite thigh. In a case of paraplegia by spinal compression—this is also an observation of Erb,—the knee sign was absent in proportion to the duration of the paralysis. It reappeared when the patient recovered the use of his legs. This tends to prove that the existence of the phenomenon is subordinate to the integrity of certain medullary regions.

I must further remark, that from the observations of Nothnagel, confirmed by Erb,[2] the sharp excitation of certain parts, more or less distant from the place where the knee phenomena are produced, suffices, sometimes to prevent their production. Thus pinching of the skin of the abdomen, or the intense faradisation of the opposite leg, suffices to prevent the contraction of the biceps crural. This you perceive is a phenomenon of arrest, analagous to those that may be produced at times in animals

1 Ibid. 1879.
2 Ziemssen's Handbook.

on which are studied the various conditions of activity of spinal reflexions.

But the reflex nature of the *tendon sign*, if there can be any doubt about it, is now a matter of experimentation. Already Furbinger and Schultze[1] have observed that the tendon reflexes exist normally in animals—the rabbit for example—as well as in man, and they cease to exist when the spinal cord is destroyed. And quite recently Tschirjew has repeated these experiments and he has determined with great precision the conditions of the phenomenon.[2]

The region of the cord, the integrity of which is necessary to show the knee sign in the rabbit is, exactly, included between the fifth and sixth lumbar vertebræ. When this region is destroyed the phenomenon cannot be produced. On the other hand the regions above and below that level cannot produce it.

Now it is the region above indicated, which gives origin to the sixth lumbar pair, and that furnishes, as Krause points out in his treatise on the anatomy of the rabbit, the greater part of the crural nerve. If then you cut the posterior or the anterior root of the sixth pair the reflex action ceases on both sides. It fails on one side only, if a section is made of either the anterior or posterior root on that side. Thus we see the nature of the reflex phenomenon well established; and it is unquestionably a reflex spinal function. I will add that the researches of Sachs have proven that the nerves in the structure of the tendon of the triceps are centripetal and particularly at the junction of the tendinous and fleshy parts. They are distended when the rotulian tendon is percussed, and transmit the excitation to the lumbar cord. This excitation, carried as far as the gray substance through the sensitive roots of the sixth pair, is reflected on the triceps muscle through the corresponding motor root, which is one of the chief contributors to the crural nerve. I will add that according to the observations of Berger the employment of strychnine, which renders the foot phenomenon conspicuous in

1 Centralblatt f. d. Nervenkrank, 1875.
2 Arch. f. Psych., viii, 1878.

disease, increases, under the same circumstances, the phenomenon of the knee.

There exist other tendon reflexes in all respects comparable to these we have just described, in various parts of the superior extremities; only, so far at least as I can judge by a few observations that I have made, they are scarcely visible in the normal state. On the contrary in hemiplegics, in the special conditions in which we study them, they become very manifest and acquire consequently a real interest. Thus, in a case where there exists a tendency to contracture in the arm, or where the contracture is already established, the percussion of the tendon of the biceps excites a brisk flexion of the forearm on the arm; the percussion of the tendon of the triceps provokes extension; and in the same way we can readily produce reflex movements in the hand and fingers by percussing the tendons corresponding to the muscles by which the movements are accomplished.

IV. I would have finished this subject, gentlemen, with the study of the symptoms if I did not feel constrained to exhibit the precious indications which can be given, in a clinical point of view, by the graphic analysis of muscular reflex contractions, which, in the pathological state, submit to such important modifications.

Already Tschirjew has applied the graphic method to the study of the phenomena that I have pointed out, and determined the duration of these reflex actions in healthy subjects. Recently Brissaud has re-examined these experiments, in my service, aided by Francois Frank, and the results he has obtained, particularly in hemiplegics, merit a very special notice now, on account of the knowledge we can obtain from them. It is only necessary, therefore, to look at these two tracings (Fig. 34, 35), to learn at a glance the difference that exists between the reflex contraction of the crural triceps in hemiplegia, and that of the corresponding muscles on the sound side.[1]

1 In these tracings the first line represents the variations of the excursions of the muscle during the contraction. The second line, bending towards the left, indicates the precise moment when the hammer struck the rotulian tendon. The third line, graduated, indicates the duration of the reflexion in five one-hundredths of a second A correction is necessary by subtracting from the time of the reflexion, the time lost in the transmission of the deformation of the muscle unto the registering cylinder in the caoutchou tubes of the instrument. By taking account of this correction the time of the reflexion is not more than the thirty-six thousandth of a second on the sound side, and thirty-two thousandth of a second on the contractured side.

The character of these traces concur to show the exaggeration of the reflex power of the spinal centre in the moity of the cord which corresponds to the paralysed side.

In effect, while on the sound side the reflex action takes place after a lapse of time equivalent to forty thousandths of a second, it is produced in the triceps on the paralysed side in about thirty-six thousandths of a second. Besides, the amplitude of the contrac-

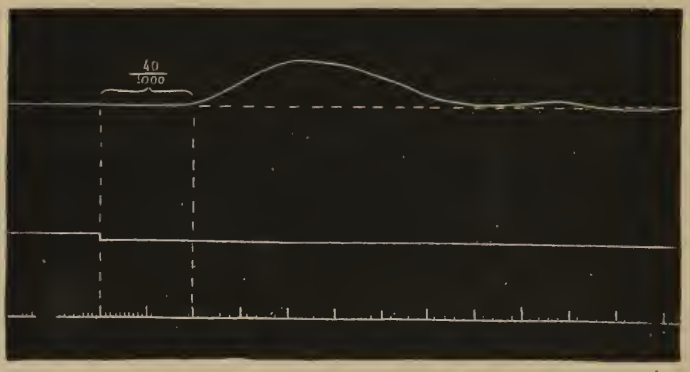

FIG. 34.—Rotulian reflexion (hemiplegia with contracture) on sound side. The time of the reflexion is forty thousandths of a second. The curve of the contraction in the normal form.

FIG. 35.—Rotulian reflexion (contractured side). The time lost in reflexion is thirty-six thousandths of a second. The muscular curve is much more elevated; the contraction is more brusque and the form is different. Dicrotism.

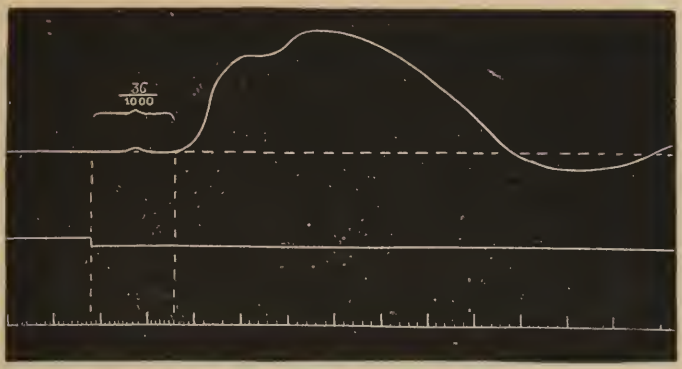

FIG. 36 —Exaggeration of the contraction of the triceps crural, following the percussion of the rotulian tendon.

tion is much greater on the diseased side than on that which is normal. That is to say the shortening of the triceps, is greater in one case than in the other and it remains longer.

Finally, the form of the muscular contraction in question, is notably different in the fact that there are nearly always successive repetitions (dycrotism or polycrotism). Sometimes, even at the end a series of oscillations more or less elevated the muscle in lieu of returning to its natural state remains for a period more curved than it was before the tendon was struck; (see fig. 36) so much so that at that time the observer sees an outline of contractures.

V. Such are the phenomena which it is important to bring out in the premonitory phase of the tardy contracture period in hemiplegia We shall find them, I repeat, with all their characters in a great number of cerebro-spinal organic affections, other than those which now especially occupy us. My object has been to make you acquainted, once for all, with their principal details.

Now I turn once more to the consideration of the prodromic period of contractures. That period may be marked by some other signs more rare than the preceding ones, and of less practical importance, but it is proper now to point them out, because they are of such a nature as to throw certain light on the question of theory, still much discussed.

A. I will speak in the first place of the phenomena known as *associated movements*, *syncinésies*, as Vulpian calls them.

As far back as 1834 Marshall Hall developed a certain number of facts of this nature, among which, nevertheless, were several which had been known before his time. It is well known that during gaping, or sneezing, there are produced occasionally in hemiplegics an automatic movement in the arm, even when it is completely paralyzed, and no voluntary movement is possible in it. But for the first time, in 1872, Onimus remarked that in circumstances analogous to those now under consideration, the occlusion of the hand on the non-paralyzed side, or the individual movement of either of the fingers, excited similar movements in the hand or fingers of the paralysed side. These movements provoked by *association* are, I repeat, entirely involuntary, and, generally speaking, it is only by a great effort of the will that patients can prevent their production. Westphal has likewise noted some curious facts which he had observed in several cases of infantile hemiplegia. But I can only refer to them in passing, for they are not of common observation and are interesting in a theoretical point of view only.

B. Equally very curious in the same point of view, but very important for practical purposes, are the effects produced in the paralyzed members of a hemiplegic stricken with contracture, by the use, particularly of nux vomica or strychnine. Already we have seen that the employment of this therapeutic agent brings on in exaggerated or other forms the tendinous reflex action. We shall now see it precipitating, in a manner, the events, and determining the appearance of the phenomena of contracture.

You must have heard of the researches of Fouquier, once a professor in this faculty, on the *use of nux vomica in the treatment of paralysis*. That is the title of the second article published on this subject by him about 1820. The first is dated 1811.[1] This is a very interesting work to consult. We find there the origin of all the information we are now possessed of on the subject we have in hand. It comprises fifteen or sixteen observations relative,

1 This second article is inserted in the fifth volume of the memoirs of the old society of the Faculty of Medicine; it has been reprinted in full in second volume of the Bibl. de Therap., published by Bayle, 1820, p. 141.

some to paraplegia by compression or myelitis, others to common hemiplegia of cerebral origin. Enlightened by the experiments of Magendie, Fouquier aimed to produce in paralytics an artificial *tetanus*, and in this way a means of cure. Among the remarks he made at the close of his observations I will note the following:

"A remarkable fact," says Fouquier, "nux vomica can produce contraction of paralysed muscles without disturbing the sound ones. Taken in common doses it only acts on the diseased parts. It seems that these exhibit much more distinctly the medical effect when they are most completely paralysed. These are facts that it is impossible for us to refer to physiological laws."

Doubtless at the epoch when this work was published these facts were judged to be incompatible with all the data of pathological physiology. But at this time, when the action of strychnine on nervous centres has been studied by the experimental processes, the interpretation of these phenomena becomes very easy.

Another remark of Fouquier is that in provoked contractions in paralysed members by the employment of nux vomica, the thoracic members become flexed, and the abdominal extended. We shall soon see the interest of that remark.

But here is the last point on which I wish especially to fix your attention. "Paralytics," says Fouquier, "and this relates to hemiplegia as well as paraplegia, conserve often after the administration of nux vomica, a considerable rigidity of the members though the use of the remedy has been for a long time suspended." Thus you see a durable contracture produced by strychnine, the same medicine which we have just shown makes so conspicuous the tendon reflexes. This is an important circumstance to mention, for it shows that tendon reflexes and contracture are connected phenomena. I shall have occasion very soon to return to this subject.

LECTURE THIRTEEN.

ON THE TARDY CONTRACTURE OF HEMIPLEGIA AND THE CLINICAL VARIETIES.

Summary:—Premature appearance of Tardy Contracture determined by Faradization and Traumatism. Example given.—Connection of Contractures of Hysteria and Hemiplegia.—Description of Hemiplegic Contractures.—Vicious Postures of Members a Resultant of Opposed Action of Antagonistic Muscles.—Paralytic Contractures.—Contractures by Adaptation.—Myopathic Contractures. Contracture of Hemiplegia is a state of Muscular Activity.—Compared with Normal Contraction.—Limits of Voluntary Muscular Contration.—Muscular Tonus comparable to Permanent Contracture of Sphincters.—Chemical modifications of the Blood which passes through Muscles.—Theory of Onimus on indefinite Contraction.—The Muscular Sound investigated with a Telephone by Brissaud.—Vulpian's views of the influence of Spinal Centres on Muscular Tonus.—Permanent Contracture amends during Sleep and Horizontal Repose.—Postures of the Limbs in Chronic Hemiplegia.—They are conformable to Certain Types.—Examples in the Superior and Inferior Members.—Contracture as seen in Muscles. How Contractures Terminate.

GENTLEMEN:

I reproduced, in terminating my last lecture, the various influences, which, in durable hemiplegia of cerebral origin, may augment the intensity of the phenomena known as tendon reflexes. In regard to the effects of strychnine I showed you that this agent provokes, equally, under the same circumstances, the appearance of tardy contracture, then in a manner, imminent;—that there is, in short, a preparation for its production.

This is a fact worthy of notice because it seems to indicate that tardy contracture, and the excitation of tendon reflexes are connected phenomena, and related to a common condition.

I wish now to remark again, that other causes, which apparently have nothing in common with the therapeutical uses of strychnine or some other medicines may, nevertheless, determine also pre-

maturely, the appearance of contracture in hemiplegia; or render it more intense when it is already established.

I. First. We have known for a long time in electrotherapy that inordinate and badly applied faradisation of the extremities in patients of this character may provoke, or exaggerate permanent contractures. But it is less known that a traumatism of the paralysed members may also be cited among the causes of this nature. As an appropriate example to exhibit the reality of this fact, I will mention the following case recently observed in the hospital of Salpétriere by my colleague, M. Terrier, who at once saw its therapeutical interest and has therefore reported it with great care.[1]

It relates to a woman, aged 52, who was suddenly stricken with a hemiplegia six years ago, in consequence, very probably, of a partial softening. The hemiplegia has persisted to a certain extent since that event, but it has only been revealed by a slight rigidity of the right superior member; the inferior member was almost unaffected; she walked easily and her exercise in that way was somewhat lengthy. On the 29th of last March she fell from a seat, but her legs being flexed the only injury that followed, was a contusion of the front of one leg which on the next day was swollen and ecchymotic. But observe this interesting point; the day after the accident the right inferior member, that is, the one corresponding to the paralysis, became rigid generally; it could only be raised as one mass; the foot presented the posture of equino-varus; in short, it was a true contracture, as characteristic as possible. Moreover the contracture has increased considerably in the superior member where before there was only a slight flexed contracture, rendering the spontaneous or provoked movements very painful. To-day, six weeks after the traumatism, the contracture of the members is improved, but it persists still to quite a high degree; and the foot phenomenon and the rotulian reflexion are greatly exaggerated in the inferior member.

2. Facts of this nature are not, doubtless, very rare; I have collected two or three. On this subject it will be interesting, I think, to establish a connection, in the special point of view that

1 Revue Mensuelle de médecine et de Chirurgie, 1879, No. 12.

we have now in hand, between the permanent hemiplegia, resulting from an organic cerebral lesion, and the hemiplegia, that reveals no appreciable alteration, which is sometimes seen in hysteria. It is known that in hysteria with hemianæsthesia, especially, the members on the insensitive side are frequently attacked with paresis more or less pronounced. This paresis may become aggravated to the point of complete, veritable, flaccid paralysis. I have observed that the tendinous reflexions are in general manifestly exaggerated in the members thus paralysed, and so far as the inferior member is concerned, there an epileptoid trepidation may be provoked.

In such a case, it has several times happened to me to determine, in the members stricken with this impotency, the appearance of a contracture more or less intense and permanent by the application of feeble galvanic currents, or even by the simple application of a magnet. This result enables us possibly, to comprehend why contracture is very often developed so suddenly, in certain nervous subjects, as a consequence of the commonest injury. I have recently called attention to these singular facts which were already known to Brodie. Thus, following a fall on the wrist, or a rather strong pressure on the back of the foot, I have seen developed, in certain subjects, almost immediately, in the corresponding member, a contracture which continued in a permanent state during several weeks and even months. The appearance of the contracture thus produced, is often the first revelation of the hysterical diathesis. But, on closer inspection, it is nearly always seen that on the side where the contracture is developed, there exists an anæsthesia more or less distinct, an ovarian pain, a certain degree of paresis, —conditions relatively benign, but which all lead to the belief that they have preceded the appearance of contracture. The foundation was thus in a manner already laid and the traumatism has played the part, simply, of a provocative agent.

I limit myself, for the moment, to indicate the connection between the contractures of hysteria and the contractures of hemiplegia. I shall have occasion, certainly, to profit by this hereafter.

II. It is now time to return to the description of hemiplegic contracture, of which I have thus far given you only a summary.

In ordinary circumstances, that is when it supervenes spontaneously, it is but slightly established until about the middle of the second month, sometimes later, rarely earlier; it is exceptional for it to be manifested as early as twenty days after the attack, as Vulpian has seen it in one of his patients.

Be that as it may, it is not sudden but comes on gradually. It is rare moreover for patients to inform us exactly in this regard. But if you have occasion to watch one of these cases in this period of transition, you will observe that before the time of its definite installation the contracture appears from time to time in a fugitive manner. On one day you will see it, then it disappears, but will soon come again. Finally it is established. In the immense majority of cases it is the upper extremity in which it is first localized. The fingers are more or less flexed on the palm of the hand, the elbow is in demiflexion and the fore arm is in pronation.

Besides, as an effect, it produces distorted postures in the members, nearly always the same, and I shall give you directly a brief description of them, for it is interesting to remark that these deformities are not accidental but are subject to a law.

Meanwhile, it is important, first of all, to consider contracture itself and to determine accurately its character in hemiplegia; considering that we shall see the same phenomenon with all the particulars we shall describe, in a large number of spinal affections other than those which now occupy us, and it is, as I shall proceed to show you, gentlemen, a strange phenomenon, paradoxical to a certain degree, and very difficult to interpret in every case with the present data of physiology.

Please remark that these immobile members, in a condition of distortion, thenceforth nearly definite, are in such a state that the observer who wishes to move them will find a resistance more or less pronounced and sometimes nearly invincible, in whatever way he attempts to displace them. Thus, to speak of the elbow which, we have said, is in demiflexion; it is just as difficult to increase the flexion, as the extension. There is about the same resistance in both orders of muscles, which will convince you that these antagonistic organs are contractured in about the same degree.

These vicious postures represent then the resultant of the opposed action of the antagonistic muscles. If the biceps is stretched like a cord the triceps is equally hard and rigid; and could we overcome for a time the resistance in one direction or the other, the arm left to itself, would return almost at once to its primitive posture.

B. 1. This fact suffices therefore to show, that the phenomenon is quite other than that which produces the deformities common to certain paralyses, and which sometimes are designated as *paralytic contractures*, *contractures by adaptation* (Dally). The deviations which are seen in infantile paralysis (at least in its early phases and when the phenomenon has not yet been disturbed by diverse circumstances which may supervene ulteriorly) offer the most characteristic type of so-called paralytic contractures. Let us suppose that it concerns an atrophic paralysis of the muscles which, normally, produce the dorsal flexion of the foot. The muscular tonicity in a manner incessant, and the first efforts of the patient in a manner intermittent, exercise, exclusively, the gastrocnemii muscles, the activity of which produce a plantar flexion, and the predominance of the action of these muscles will bring on at length the posture of talipes equinus. But it is always easy when the case is not of too long standing to re-establish, momentarily, the normal attitude without resistance on the part of the paralysed muscles, which are, for the time, deprived both of tonicity and of voluntary motion.

2. It would be equally easy to distinguish the permanent contracture of hemiplegia from contractures called *myopathic;* that is, those in which there is a lesion of the muscular tissue itself, as cirrhosis for example, as well as that which is seen in certain facial paralyses. In tardy contracture, on the contrary, at least when it is not of very long standing there is (autopsies have many times demonstrated this) no alteration of the muscular tissue, and when this is produced, generally after quite a long time, it consists in simple emaciation. During life the faradic excitation reveals also, in the contractured muscles, a normal excitability; sometimes even more than that. To sum up, gentlemen, the contracture of hemiplegia is not a passive rigidity. It responds on the contrary to a

state of muscular activity. It is, doubtless, a phenomenon comparable to a normal contraction; only it is a durable, permanent contraction. This persistance, indeed, of the activity of the muscles constitutes, justly, the paradoxical character which we mentioned just now. Day and night, in fact, during months and years, these muscles remain rigid, and sometimes in postures which an effort of the will in the normal state could maintain for a few moments only.

According to the experiments of Gaillard (de Poitiers) one cannot hold the arm, in ordinary circumstances, in a horizontal position longer than nineteen minutes. The most robust person was not able to stand tiptoe by the contraction of the gastrocnemii muscles more than three minutes. On the contrary, the contracture in question maintains the inferior members, indefinitely, in forced attitudes and often in a violent manner.

The permanent character of contracture appears not less singular when we consider the intensity of the chemical phenomena of nutrition which occurs in a muscle during the act of contraction.

3. There exists, however, a normal phenomenon which, without going too far in analogies, seems to be comparable to the permanent contractures of hemiplegia. I refer to tonicity or the muscular *tonus*.

You are aware, gentlemen, that certain muscles, the sphincters for example, are manifestly in a state of permanent contraction, which is also the state, though in a less degree, of all the muscles of animal life.

These muscles, in the state of so-called repose are I repeat, in a state of incessant, active contraction, and which only disappears when the corresponding motor nerve has been sectioned. The tonic contraction of the muscle is revealed also, as C. Bernard has proven, by a chemical modification of the blood which passes through the muscle. Thus the quantity of oxygen contained in the arterial blood which penetrates the muscle being represented by 7.31 *per cent.*; the quantity contained in the venous blood, as it issues from the muscle during contraction, amounts only to 4.28 *per cent.* When the motor nerve is cut the tonicity of the muscle is abolished and the quantity of the oxygen in the venous blood

is then about equal to that in the arterial blood. But in the muscles in a state of repose, or, in other terms, in a state of simple tonicity, the nerve being intact, there is in the venous blood but 5 *per cent.* That shows clearly that the consumption of oxygen in tonicity represents nearly a third of the total quantity contained in the blood of the afferent vessels. I will remind you that to explain this apparently paradoxical fact, of permanent, indefinite contraction of the muscle, Onimus has proposed that it is due to the contractions taking place successively, and not simultaneously, in the diverse fasciculi of the muscles, and in such a manner that a part are in repose while the rest are in contraction.

This hypothesis of Onimus' has been sanctioned also by the experiments which Boudet and Brissaud have recently made, in my service at Salpétrière, on the muscular bruit. By the aid of a microphonical apparatus for auscultation, of exquisite delicacy, the analysis of the muscular bruits in the normal and pathological state have been carried as far as possible; and I will briefly give the results that this new method of investigation has furnished, in the special point of view of our present study. Whilst the muscle in normal contraction produces a regular, sonorous, rolling sound (bruit rotatoire) constant in the number of its vibrations; the contractured muscle produces only a dull, heavy, irregular sound, with the interruptions and resumptions, or, in other terms, intermittent in character. It seems, then, patent, that here the muscular fibres follow each other in contraction, and relieve each other unceasingly.

I must add that according to the teachings of physiology, the slight but permanent contraction of the muscles, called the muscular tonus, depends upon a constant stimulation that is exerted by the spinal nervous centres. "The spinal cord," says Vulpian, "acts incessantly upon all the muscles in which it produces through the motor nerves the muscular tonus. This continuous action of the cord is excited, doubtless, by the centripetal excito-motor stimulation proceeding from the muscles themselves, or the integuments." It is therefore a phenomenon which reveals the reflex action of the spinal cord.

Now, it would be very easy for us to bring forward a certain

number of facts which tend to prove that the permanent contraction of muscles in the case of contracture is due to an analogous origin; that is, derived also from a permanent spinal action exalted by certain pathological conditions. Thus the phenomena which precede and accompany the development of contracture are due, as you may easily suppose, to an exaggeration of spinal activity. The employment of strychnine which brings about the tendon reflexes, provokes in a like manner, the appearance of contractures, or exaggerates them when they already exist; and, inversely, the agents which depress the activity of spinal reflexion diminish equally the intensity of these contractures. It is in this way the bromide of potash acts when given in high doses.

But I will not dwell longer on physiological considerations, as I will recall them by and by; and I resume again the descriptive part.

III. A. Contracture is said to be permanent; nevertheless, in reality it amends naturally in most patients without, however, completely disappearing, during sleep and rest in bed. It resumes, on the other hand, its intensity or grows worse, as Hitzig was correct in showing, during emotional excitement, or when the patient rises or attempts to execute any movements. We will see how it has been attempted to explain this phenomenon which has been placed by Hitzig in the category of associated movements.

B. A few words more in regard to the postures of members in chronic hemiplegia. It is very remarkable to notice that these postures are conformable as a rule to a fundamental type. In the superior members flexion predominates, while in the inferior members extension is the rule. Fouquier had already shown that the same phenomenon is produced when temporary contracture is developed in hemiplegia by the use of strychnine. I will remark that the same thing is seen in partial epilepsy.

1. Let us take first the superior member. Here we have the flexion type. Bouchard has observed this attitude in twenty-six out of thirty cases. The shoulder is sometimes depressed, at others elevated; but the arm in all cases is pressed to the side of the thorax by the contracture of the pectoral muscle. We have

seen, above, that the elbow is usually demiflexed, the forearm is in pronation and the hand firmly closed.

All the varieties of the type are embraced in the following: (*a*) the elbow resting flexed, the forearm is in supination. This is the type of flexion with supination. (*b*) The elbow is in extension. The forearm is more or less extended. This is rather a rare type and besides presents several varieties; (*c*) sometimes the fore arm is in pronation; (*d*) again it is in supination. I do not think that so far as the superior member is concerned, there are any other postures than those above mentioned. The open hand is a rare fact.

2. In regard to the inferior member, the rule is, as I have said to you, for it to be rigid in extension. The foot therefore takes the posture of talipes equino-varus. Other things being therefore equal, the contracture is habitually more pronounced than in the superior member. A marked degree of extension in the lower extremity does not prevent walking, but in the mowing style (*en fauchant*).

The development of contracture in the flexor group of muscles in the leg is a very unfortunate condition but is, happily, very rare.

In such a case the thigh is flexed on the pelvis, the leg on the thigh, the heel touches the breech, and when this flexion extends, as it sometimes does, to the opposite limb, it is plain that walking is, henceforth, impossible.

3. Finally, it is not exceptional that contracture of the facial muscles is established on the paralysed side in the lower region, of course. This contracture at first, is occasional, as when the patient laughs or cries, but at length it becomes permanent. The labial commissure is very elevated, the naso-labial furrow is deeper and the eye sometimes appears much smaller than its fellow on the sound side. By contrast, it is then the sound side that seems to be paralysed, and at first sight it may be taken for an alternate paralysis.

4. I have presented the tardy contracture of hemiplegia as a particular state of muscles which, when once established, remains throughout life, or at least for many years. But it happens often that the muscles are destroyed by suffering and wasting. Thereafter the muscular spasm ceases and exactly speaking there is no

more *contracture.* Nevertheless the long continued postures of members may still persist on account of the shortening of the ligamentary parts, so that the articular surfaces have become accommodated to their new situation and voluntary movements, if they reappear at all, must henceforth of necessity be very limited.

Is this termination the only one possible? Does the contracture never disappear before the time when the ligamentous contraction and muscular atrophy have taken place? Some authors think so. At all events, it is certain that there supervenes sometimes a notable amendment in contracture, and on this account the voluntary movements may be reproduced. That is what may be called a cure, but truly a relative one. They are unhappily rare. I have seen several of them and doubtless some modification had supervened in the diseased parts, permitting not only the disappearance of the contractures, but also the return of motility to a certain extent.

Does the lesion persist in the spinal cord, and in such a case is there a metonymy? How then does it act? Or has there been brought about something like a reparation of the nervous elements within the indurated pyramidal fasciculus? This is, certainly, a very interesting question, but still very obscure, and about which I shall tell you something when we consider permanent contractures of spinal cause, where the same phenomena are produced.

LECTURE FOURTEEN.

SPASMODIC HEMIPLEGIA OF INFANCY; ASSOCIATED MOVEMENTS; INDEPENDENCE OF DIASTALTIC ARCS FOR THE TENDINOUS AND CUTANEOUS REFLEXIONS.

Summary.—Hemiplegia of Young Children—Infantile Spasmodic Hemiplegia.—Other Points in Permanent Contracture.—Modification in Contraction during Rest and Sleep.—Seguin and Hitzig on the Effect of Voluntary Movements.—Hitzig's Explanation of the Phenomena as related to the Structure of the Cord.—Charcot's objections.—How Contracture Terminates.—Charcot's Hypothesis and Description of Contracture: It never exists in the New-born.—Contracture arises from Irritative Lesions of the Pyramidal Fasciculus, and Involvement of Ganglionic Cells.—Analogy of Contracture and Muscular Tonus.—Spinal Reflection, Cutaneous, Tendinous and Muscular Reflexions represented by two Diastaltic Systems.—Examples in Hysteria, Ataxia and Traumatism.—Diagnosis illustrating the Theory.—Permanent Contracture not a Function of Sclerosis of the Pyramidal Fasciculi.

GENTLEMEN:

I. In presenting to you at the end of the last lecture an abridged description of the diverse postures which paralysed members assume in hemiplegia with permanent contracture, I remarked that the varities, very numerous apparently, could all be included in a small number of types, always the same, viz.: a type of flexion with pronation for the superior member; a type of extension or equinism with varus for the inferior member. Such is the rule—such is the order of deformities that is observed in common cases. The other postures that we sometimes encounter represent anomalies—exceptions.

1. The law which we have just announced as apposite to the permanent hemiplegia of adults is reproduced in the durable hemiplegia of young children. You are doubtless aware that in children from one to seven years, focal lesions of a diverse nature—when they involve the intra-cerebral tract of the pyramidal

fasciculus—are followed by a hemiplegia more or less pronounced, and which persists, to a certain degree during life. The causal lesion of the hemiplegia is, as has been shown by Cotard, at that time my interne, of a varied nature.[1]

Sometimes it is due to a partial softening, occurring in the form of a yellow plate, or a focus of cellular inflammation; at another, the lesion is consecutive to a meningeal hæmorrhage; again, and this is most frequently the case, it is due to a partial or generalized sclerosis of one of the cerebral hemispheres. These lesions are usually cortical, that is, they occupy the mantle and not the central masses. Also, the affected cerebral hemisphere is in a condition of atrophy, more or less marked, whence the denomination *partial atrophy of the brain*, under which is generally ranged the cases that I have brought to your attention. Secondary descending degenerations are there seen with all the characters by which they are recognized in adults; and it is under these circumstances that we see the most beautiful examples of atrophy of the peduncles, the protuberance and the bulbar pyramid, on the side corresponding with the lesion.[2]

Clinically, the facts of this order are sometimes designated as *infantile spasmodic hemiplegia* (Heine). It is, in fact, permanent contracture in strong colors. The deformities are, besides, related, as I have previously announced, to the type described as pertinent to hemiplegia of the adult. Thus, for the superior member the flexion type with pronation is here also the rule; and for the inferior member it is the extension type, in equino-varus.

However, in the history of infantile spasmodic hemiplegia a very interesting peculiarity must be mentioned, viz.: the almost constant condition of shortening of the paralysed members. The bones are shorter and less voluminous than on the sound side; and this arrest of development does not always bear equally on the members; thus, sometimes, on the paralysed side the trunk is incompletely developed—the thoracic cage is narrow and the pelvis is shrunken and oblique. This is the deformity that atrophic paralysis, resulting from a lesion of the spinal gray substance,

1. On the Partial Atrophy of the Brain. 1868.
2. See observations published by Bourneville in Progrés Medical, No. 16. April, 1879.

induces when it exists in infancy; a shortening by arrest of development of the member, in which are seated the muscular lesions, even when these members fulfil in part their functions; while this shortening would not naturally exist when the same lesion is developed in the adult.

2. In the description, though greatly detailed, that I have given of permanent contracture in hemiplegia, there are yet some points which I have neglected to bring out, and about which I must beg your attention for a short time. One of these, especially, should be signalized because some authors have accorded a great importance to it, in a theoretical point of view.

I have presented to you the tardy contracture of hemiplegia, as being a permanent phenomenon, in the rigorous acceptation of that word. Night and day, I have said, in sleep or awake the members are rigid and contractured. This is really the rule, at least in well marked cases. Nevertheless, it is certain that during rest in bed and in sleep, there may be a brief period of flexibility in the members; but as soon as the patient rises, or attempts to execute any movement, with either the stricken, or sound member, at once the contracture reappears with all its intensity. This augmentation and return of rigidity under the influence of voluntary excitation, is especially brought out, as Seguin and Hitzig suggest, when, the patient, being contractured on the right side, is asked to raise a weight with his left hand. The heavier the weight the more the contracture is exaggerated on the right side. These facts are considered, partly, as belonging to the category of *syncineses* or associated movements, about which I have already made some remarks.

See, moreover, how Hitzig proposes to explain these phenomena to which I have called your attention. In the normal state the voluntary excitations, parting from the gray substance of the hemispheres, are conducted through the cord by the nervous fibres which place them in direct relation with groups of cells which have particular connections among themselves; and these are the cells which execute the purposed movement. There exist there also, elementary groups for elementary movements, and associated groups for movements more complicated and com-

bined. These groups are distributed on each side of the cord, and severally preside over the movements of both sides. However, through the intermedium of the reticulum of the gray substance, relations are established on both sides, between these homologous groups. In the normal state these connections do not interfere with the independence and individuality of voluntary movements; but in certain pathological states, when the ganglionary elements are super-excitable, the least disturbance produced on one side, and which determines there a voluntary movement, may be communicated to the other side and provoke, according to the case, either movements similar to a voluntary one, or a spasmodic movement, which is really a contracture, and which persists for sometime after the disturbance.

In certain cases, relations of the same nature may be established among cellular groups, quite distant from each other, and we can comprehend that in these cases the voluntary movements executed by the sound side may be re-echoed in that which is diseased.

The facts brought forward by Hitzig in the interesting memoir to which I have already refered, and to which I shall return again, are exact; but the part which is attributed to them in contracture itself, I believe to be exaggerated, and it is necessary to consider as exceptional the cases in which the contractured members in hemiplegia, present, during rest, a complete relaxation. Hitzig has imagined a mechanism which may explain why contracture is aggravated under the influence of voluntary movements, but he does not make us understand why this contracture, as it exists in the majority of cases, is a permanent condition.

3. It is necessary, finally, to endeavor to ascertain what at last becomes of contracture. Often, very often, when once established, it persists throughout life. However, a number of cases may be referred to, where, at length, it was greatly ameliorated, and even ceased to exist. Generally, it is without great advantage to the unfortunate sufferers; for though the spasmodic state has disappeared, the muscles have undergone a modification, more or less profound in their texture, and are extremely emaciated. Also, the ligamentous parts have become adapted to the situation created by a posture kept so long fixed, and, in fine,

notwithstanding the possible return of some voluntary movement, the deformity persists.

II. Now, gentlemen, by way of conclusion, I propose to seek, with you, a physiological explanation of the phenomena which we have, thus far, considered on the descriptive side. It is important, in a word, to ascertain in what way the symptoms are connected with the lesions. This is always a delicate undertaking, and in this particular instance, where, for the questions which arise, we shall not be able, for want of sufficient data, to offer a definite solution. You should consequently consider the greater part of the explanations which I propose to offer you, as eminently provisional, and which are certain to be modified some day or other.

1. I will remind you that the consecutive lesion of the lateral fasciculi represents at its origin, according to most authorities, a purely passive process. It is in the second period only, corresponding to the second or third month, that there arises in the altered pyramidal fasciculus, the evident marks of an irritative process, of the connective tissue and which justifies the denomination of *sclerosis*.

(*a*) In the first place the nervous tubes being separated from their trophic centres, which at the same time are their centres of functional excitation, the condition is nearly equivalent, in very marked cases, to a section of the pyramidal fasciculus. This first phase, which corresponds to the first four or five weeks, is already marked, as you know by an exaltation of the cutaneous and tendinous reflex phenomena. Here we could invoke, strictly speaking, the restraining influence of cerebral action, the want of which, in the case of an experimental section of the lateral fasciculus, seems to explain the exaggeration of the reflex properties in the portions of the spinal cord situated below the section.

But this condition is evidently insufficient to account for the contracture. Contracture does not exist in the new-born, for you know, that the pyramidal fasciculi are not yet developed; it is necessary, then, to look further.

(*b*) For the rest, contracture is never seen but at a period when the pyramidal fasciculus has already become the seat of irritative

lesions. I will remind you now of the anatomical connections established between the extremities of the nervous fibres of the pyramidal fasciculus and the motor cells of the corresponding cornu. These connections are such, that in certain cases the lesion of the nervous tubes is propagated to the ganglionary cells, which become atrophied, and to the neighboring connective tissue. There is produced thus a sort of *anterior poliomyelitis*, and, consequently, a muscular atrophy supervenes in the paralysed members.

2. But these cases, you know, though far from being exceptional, nevertheless do not constitute the rule. Ordinarily things do not go so far. It is necessary to suppose—and here I ask of you the first concession—that owing to the irritation of which the nervous tubes in process of destruction are the seat, the cellular elements (ganglionary cells) become in turn affected. Now, this lesion communicated to the motor cells would be purely dynamic; it does not correspond to any appreciable anatomical modification; this lesion, if you will allow it, we will qualify as "irritation;" and it is analogous to what is produced by strychnine, but more durable. The properties of the ganglionary elements thus modified not only do not become extinct, but rather are exalted; and in this way irritation is radiated to a certain distance by the route of the nervous reticulum into the ganglionary elements of the same region, and in particular to the æsthesodic cells. An exaggeration of the reflex power in all its modes, in the parts corresponding to the gray axis would be naturally the consequence of this super-excitability of the ganglionary elements, and should furnish us the key to certain phenomena, such as the exaltation of the cutaneous and tendinous reflexions. Without pressing this view too far, we may even admit, that the irritative lesion, now in question, provokes equally an exaltation of that mode of spinal reflex activity, which, in the normal state, maintains the permanent muscular contraction known in physiology as muscular *tonus*.

III. It is not unimportant to remark, gentlemen—for it is a fact of great practical interest—that the two modes of spinal reflex activity, now under notice, are, reasonably, represented in the

gray substance by two distinct *diastaltic systems*. Clinical observation demonstrates, indeed, that though these two modes of reflex activity are often simultaneously affected, nevertheless they may be also, frequently, separately affected. Here are some examples, tending to prove that this is really so.

1. In locomotive ataxia the cutaneous reflexions persist the most frequently, and are even, sometimes, manifestly exalted, but

Fig. 37.—*Tendinous and cutaneous diastaltic arcs in locomotor ataxia.* A, motor cell in the spinal cord; E E, æsthesodic cells; M, the muscle; P, the skin; R, posterior, or centripital segment of the diastaltic arc; P, A, M, intact cutaneous diastaltic arc in ataxia; K, anterior root of the motor nerve; T, centripetal, or posterior segment of the diastaltic arc; M, A, M, tendinous, or muscular diastaltic arc affected in ataxia.

the tendon reflexions disappear very early. It is the same of the muscular tonus. The muscles consequently are flaccid, and this diminution of *tonus* contributes, certainly, in a great measure, to give to the gait, and movements of the limbs, which conserve otherwise for along time great energy, their jerky, brusque and

irregular character. The situation could be represented in such a case by the following scheme (See Fig. 37.): the diastaltic arc of cutaneous reflexions is not affected; the diastaltic arc of tendinous reflexions, and of the tonus, is, on the contrary, profoundly affected, and from the beginning. It is one of the great characteristics of the disease.

2. I can cite at least a case in which the tendon reflexions

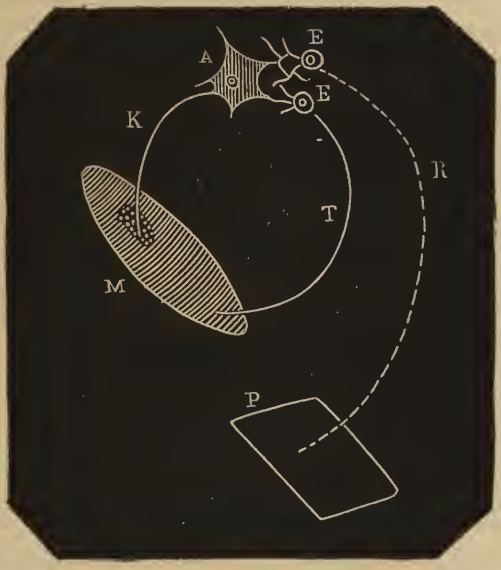

Fig. 38.—*Tendinous and cutaneous diastaltic arcs in hysteria.* A, motor cell of the spinal cord; E, E, æsthesodic cells; M, the muscle; P, the skin; R, centripital, or posterior segment of the diastaltic arc; K, anterior root, or motor nerve; P, A, M, cutaneous, or diastaltic arc affected in hysteria with hemianæsthesia, and hemiparesis; T, centripetal, or posterior segment of the diastaltic arc; M, A, M, tendinous, or muscular diastaltic arc, the properties of which are maintained, or even exaggerated in hysteria.

are exalted, while the cutaneous reflexions are abolished. It is in hysteria with hemianæsthesia and hemiparesis. The tendinous reflexions of the side corresponding to the hemianæsthesia (knee phenomenon, spinal trepidation) are very pronounced; the cutaneous sensibility is null; all excitations, even the most violent,

give no results; they are not followed by any movement. (See Fig. 38).

3. On the contrary, in durable hemiplegia, due to a focal, organic lesion, which we shall particularly study, the two diastaltic systems are affected unequally. The cutaneous reflexions are only moderately excited; the tendinous reflexions and the tonus, especially, which is a connex phenomenon, are excited to a high degree.

IV. However that may be, gentlemen, but admitting the hypothesis of irritation of the ganglionary elements of the central gray substance in contact with the extremities of the nervous fibres of the pyramidal fasciculus, and we can account for the diverse phenomena brought out in the progress of our descriptive study.

Strychnine will act, particularly on the paralysed members; its action, though extending throughout the cord will be seen, things being otherwise equal, more intense in the parts of the gray axis previously excited.

2. The irritative influence of a traumatism of the affected members will be transmitted to the central axis either by the centripetal cutaneous nerves, or by the centripetal muscular nerves.

3. Finally, in the category of associated movements, we understand readily, the aggravation of the contracture under the influence of voluntary movements, impressed on the sound members. It is sufficient to admit here, that the irritation extends by a species of diffusion, to the ganglionary elements of the gray substance of the non-paralysed side. This same diffusion of the irritation may readily explain the cases of hemiplegia to which Déjerine has called attention, in which trepidation, or even contracture, is developed on the sound side.

The intensity of the hypothetic irritation of the ganglionary elements, whence is derived the reflex super-excitation, will appear, otherwise, variable, according to the case and the patient, as well as the extent of the regions through which it is propagated. This explains, that permanent contracture connected with consecutive sclerosis of the pyramidal fasciculi is, in this species, a contingent symptom, and not a necessary one, or pathognomonic. As a rule, it is always present; but it may easily happen that it is

absent, when, even, lateral sclerosis exists; and, inversely, that it is manifest, when lateral sclerosis does not exist, as, for example, in the contraction of hysteria. *Permanent contracture*, in other terms, *is not*, so to speak, *a function of sclerosis of the pyramidal fasciculus*. This is an important fact to understand, for the sound interpretation of a great number of facts in spinal pathology, and which we shall have, elsewhere very soon, occasion to put it to profit.

Gentlemen, it remains for me to show that the theory which I have just emitted, however imperfect it may be, is superior, nevertheless, to others which have been proposed to resolve this litigated question. The theory of encephalitis has prevailed ever. There is, in fact, no encephalitis in a case of focal cerebral hæmorrhage; at least no one wishes to give this name to the work of connective vegetation, which is carried on at the margin of the sanguine effusion. Now, hæmorrhagic foci do not produce contracture, except when they are located in such a way that they destroy the continuity of the pyramidal fasciculus; moreover, we shall see a primitive sclerosis of the pyramidal fasciculi give rise to contracture, though there exists no encephalic lesion.

It is asserted, also, that contracture results from the irritation of the medullary tubes which remain unaltered within the sclerosed parts. These tubes being quite rare and sometimes completely absent, when contracture exists, I do not exactly see how the irritation of these tubes can produce contracture. It is then necessary here again to invoke the affection of the ganglionary elements of the gray substance.

LECTURE FIFTEEN.

PHYSIOLOGICAL CHARACTER OF THE PYRAMIDAL FASCICULUS IN PERMANENT CONTRACTURE; HEMIPLEGIA; MYELITIS FROM COMPRESSION; SPASMODIC DORSAL TABES.

Summary:—Review of the Theory of Systematic Lesion of the Anterior Cornua.—Muscular Tonus.—Reflex Action related to Two Diastaltic Systems in the Gray Substance.—Examples in Hysteria, Locomotor Ataxia and Hemiplegia.—Amyotrophic Lateral Sclerosis.—A new Pathogenetic Explanation of Contracture.—Is Permanent Contracture an Habitual Symptom of Sclerosis of the Pyramidal Fasciculi?—Example in Potts' Disease from Compression of the Cord.—What is the Termination of this Contracture?—Is there Regeneration?—Description of Spasmodic Paraplegia.—Its Pathological Anatomy not yet decided.

GENTLEMEN:

From certain indications, which could rarely escape a professor of long service in conflict with the difficulties of teaching, I have concluded that the theory proposed to account, physiologically, for permanent contracture in hemiplegia has not been grasped in all its details by some of my audience. I will therefore restate, in a few words, my views. It is not, by any means, that I attach an exaggerated importance to the theory, for I was the first to recognize all its imperfections. If I press it, it is because I really believe it to be superior to all those which, hitherto, have been proposed to resolve this litigated question; for, above all others, it seems to me, to facilitate the interpretation of facts that the practitioner must encounter daily in cerebro-spinal diseases.

I. 1. I recalled, first and foremost, the anatomical relations, certainly very direct, which appear to exist between the terminal extremities of the nervous fibres of the pyramidal fasciculus and the motor cells of the anterior cornua, in the whole length of the spinal cord, and I have proposed to admit that the irritative lesion

of which the degenerating fibres are the seat is reproduced in the ganglionary elements. Thus communicated to the motor cells, this lesion would be purely dynamic, and would not be accompanied with any appreciable material alteration by our means of investigation; comparable, if I may so say, to that modification which is produced in the same elements during strychnine intoxication, but with this difference always, that the lesion of strychninism is a phenomenon essentially transitory, while that whose existence I have supposed, is eminently durable, as much so as permanent contracture itself.

The irritative state being once admitted, there is good ground for believing that it is not confined to the motor ganglionary element, but that it extends by diffusion to the other ganglionic elements of the region with which it has otherwise anatomical connections more or less direct by the intermediation of what has been called since the time of Gerlach, the *nervous reticulum*. In this way the œsthesodic cells, which are supposed to be the principal abuttal of the centripetal nervous tubes, would be affected in their turn in a similar manner. Now the motor or kinesodic cells and the sensitive or œsthesodic cells, constitute the central part of the several diastaltic systems through which are effected the spinal reflex actions; and in consequence of the supposed lesion, the properties of the diverse parts of this system should become exalted. If this be true the smallest excitations coming from the periphery in reacting on the central parts of the diastaltic arcs would be translated into reflex phenomena, more energetic than ordinary. In this manner the permanent incitations emanating in the normal state from the muscles or their aponeuroses, by way of the centripetal nerves, determine the phenomenon of incessant reflex activity known as *tonus*, and of which the physiological expression is a slight and permanent muscular contraction. In the pathological state these same incitations are revealed by a contraction equally permanent, but very intense, representing in a certain fashion the muscular tonus pushed to its supreme power: and such would be in sum, the cause of the permanent contracture in hemiplegia.

2. I will remind you how, afterwards, basing myself on consider-

ations of a clinical order, I was led to remark to you, that the diverse forms of spinal reflex activity, discerned by pathological analysis, appear to be represented in the gray substance of the spinal cord by so many distinct diastaltic systems. Observation demonstrates, indeed, that if the diverse forms of reflex activity may be effected sometimes simultaneously and to the same degree, they may be also lesed separately. Recall the particular cases of which I have made mention; hysteria and locomotor ataxia.

On the contrary in hemiplegia, which we have specially considered, the two systems appear stricken in a manner about equal. Nevertheless it occurs, according to some observations made by Rossenbach,[1] that in the first stages that follow the attack, after the period where all the reflexions may be momentarily abolished, that the tendinous reflexions appear very much earlier than the cutaneous ones. Certain among them, more especially, are remarkable in this regard; for example, that of the cremaster, produced by the action of a cold substance to the thigh of the corresponding side[2]; or yet, the reflexion determined by the excitation of the skin of the abdomen on the paralyzed side. (*Bauchreflexe de Rossenbach.*)

Continued researches made in the directions that I have just indicated would furnish, very probably, interesting data relative to diagnosis and prognosis in a certain number of cerebro-spinal diseases.

3. In the last place, I will recall also some examples that I have invoked to show that the clinical phenomena brought out during our descriptive study of permanent contractures find an easy interpretation by the proposed hypothesis.

(*a*) All the ganglionary elements of the spinal gray substance are doubtless affected simultaneously by strychnine introduced into the circulation. But those, naturally, react first, which are the most excitable. The influence of traumatism in the development of contracture is explained in very nearly the same fashion.

(*b*) If we suppose that through the commissures, the ganglionary

1 Arch f. Psych., iv, Bd. S. 845.
2 Jastrowitz, Berlin Kl. Wochenschrift, 1875.

irritation is diffused from one anterior gray cornu to the other, we will comprehend how a voluntary movement which excites the gray substance of the left cornu, for example, reacts on the right cornu and determines there either an involuntary homologous movement, or an aggravation of the contracture. We will comprehend also why the exaltation of the reflexions and even of permanent contracture can be produced at any moment on the sound side.

II. At that point we stopped. There remains still to show you that our theory is superior to all others that have been proposed.

1. The old hypothesis of an encephalitis developed around the focus is untenable. There is no other encephalitis surrounding apoplectic foci than the conjunctive, vegetative process which in the end forms the cicatrix; and on the other hand, the foci never determine permanent contracture, except, when, on account of their location they interrupt the course of the fibres of the pyramidal fasciculus; which brings on secondary degeneration.

Elsewhere we were able to cite at least one example of a primary lesion affecting, systematically, the pyramidal fasciculi, and independently of any focal cerebral lesion, or of encephalitis.

It concerned an affection that I have several times already studied with you, and of which I wish only to detach to-day an episode. I have proposed to call, *amyotrophic lateral sclerosis*, that disease in which the two systems of the pyramidal fasciculi are affected both in the cord and in the bulb. Only the lesion cannot be followed upwards ordinarily beyond the cerebral peduncle; it seems then to progress from below upwards[1]. The alteration reacts on the gray substance of the anterior cornua of the cord and on the analogous gray parts of the rachidian bulb; and it follows two methods. In certain regions it is a destructive lesion of the cellular elements. The consequence is then, an atrophy of the muscles which are in relation with the nerves emanating from the diseased gray substance. In other parts it is a simple irritative functional lesion of the ganglionary elements. As a result, in the parts, besides the paralysis more or less pronounced, there is a

1 Some facts recently observed establish, nevertheless, the possibility of intra-encephalic lesions of the pyramidal fasciculi. See, Charcot, Conférence de la Salpetrière in Progrés Médical, 1880, No. 3.

notable exaggeration of the tendinous reflexions, and even, at a certain moment, a considerable contracture, occasionally, of the members. The contracture, or in its absence the exaggeration of the tendinous and muscular reflexions, distinguishes, clinically, according to my observation, this form of spinal muscular atrophy from that in which the cellular elements are destroyed, without any participation of the white fasciculi. I will not dwell more at length on this subject; it suffices for me to point out that the proposed theory finds here a brilliant confirmation.

2. Another pathogenetic explication of permanent contracture is as follows: it has been supposed that it results from the irritation of nervous tubes not appertaining to the pyramidal fasciculus, but which are mingled with its fibres. These nervous tubes not being separated from their trophic centres do not degenerate, but remain simply irritated within the sclerosed parts. I will remark that tubes of this order are rare, in the structure of the pyramidal fasciculus; that sometimes one cannot find a single sound tube in the sclerosed parts, and, moreover, if we suppose that these tubes might appertain to the system of short commisural fibres, they could not take a part in the production of contracture without the participation of the gray substance.

III. Please to consider, gentlemen, that in the proposed theory, and now I will finish the subject, the immediate cause of contracture is in the gray substance and not in the lateral fasciculus itself. It concerns then a deuteropathic, consecutive lesion, contingent in a manner, the degree of which may vary according to the patient, the period of life and of which the existence even is not, in the species, an absolutely necessary fact.

This is an important remark, for it enables us to comprehend that permanent contracture, though it be attached to a primitive or consecutive pyramidal sclerosis quite closely, is not, of it, however, a necessary, pathognomonic symptom. Thus, though sclerosis and contracture are, observed, in general, simultaneously, we may see sclerosis without contracture and contracture without sclerosis, as, for example, in hysteria. We can conceive, indeed, that the ganglionary irritation which pro-

vokes contracture may set up primarily, or in consequence of other lesions, than those of the lateral fasciculi. The fact does not appear to me to have been demonstrated, but I consider it as very possible. In fine, gentlemen, I purposely repeat it, the condition may be expressed in a word: permanent contracture is not a function of the pyramidal fasciculus.

IV. It is not less certain though, that whenever in spinal pathology sclerosis of the pyramidal fasciculi exists, and in whatever manner, permanent contracture figures among the habitual symptoms.

1. Let us take the case of consecutive descending degenerations of spinal cause, and let it be in Pott's disease with compression of the spinal cord. Let us consider exclusively the motor troubles which often open the scene. In such a case we do not have a blunt, sudden onset, and clearly defined as in apoplexy; the paralytic symptoms are developed, usually, slowly and progressively

Be that as it may, at a certain moment, a sense of paresis is felt, then at length a veritable paralysis, the result evidently of the interruption of the conductors of motor incitation in the antero-lateral columns, and more particularly in the pyramidal fasciculi. Remember, that the paralysis in question has nothing in common of a paralysis with contracture.

But at the end of some days or weeks the scene changes: (*a*) shocks and cramps are felt, accompanied with a temporary rigidity, recalling the corresponding phenomena observed in hemiplegia; (*b*) also, beforehand, the tendinous reflexions of the knee, etc., were assuredly much more pronounced than in the unilateral encephalic lesions; (*c*) it is the same in other modes of reflex activity. It is indeed in cases of spinal compression that the reflex movements are the most intense; they are then, at times, comparable to what we see in strychninized frogs; the acts of micturition or defecation, the introduction of a catheter determine energetic shocks and convulsive movements in the paralysed members. (*d*) Finally, sooner or later, contracture is developed and it is quite rare for it to be completely absent, unless it be a question of a particular locality, for example, when the compression is

exerted on the most inferior portion of the lumbar enlargement of the cord. As a rule, it is a contracture in extension, that is produced; and, nevertheless, it is not rare to see the inferior members drawn up to the pelvis in a forced flexion; it seems, even, that this attitude may be more common in myelitis by compression than in spontaneous transverse myelitis.

It is not unimportant to mention that all these phenomena occupy a single member when the compression is unilateral, and, naturally, it is the one which responds to the side in which the lesion is located.

But, in general, the spasmodic symptoms such as epileptic trepidation and contracture are greatly more pronounced than when it concerns a total transverse lesion.

2. What becomes of this contracture? By and by the patient grows weaker, eschars form, hectic fever supervenes, and, simultaneously, the reflex power and the contracture disappear. Sometimes, on the contrary, a progressive amelioration allows us to hope for a happier issue, and in a certain number of cases a complete and absolute cure has been observed. The spasmodic state vanishes and voluntary movements reappear; only, the members remain somewhat stiff, due to the tendinous retraction, which is amenable by surgery. Of this favorable mode of termination, Bouchard has related five cases. I have seen six or seven likewise.

To explain the anatomical conditions of these nearly hopeless cases, microscopical examinations have not yet been sufficiently numerous to enable us to speak precisely; nevertheless, in a case in my service, and studied by Michaud, everything seemed to indicate that there had been a veritable regeneration.

But I cannot, on this point, fail to remark that results of experiment favor very little the idea of regeneration, and to speak only of those the most recent, I will remind you that Eichorst and Naunyn having thought they had seen this regeneration in dogs, Scheiffendecker sought to verify their observations on the dogs operated on by Goltz. These animals were numerous and some among them had survived the operation ten, twelve and even fifteen months. Now even in these conditions, as favorable as

possible, Schieffendecker could not discover any trace of regeneration in the inferior segment of the cord; the fibrillary cicatrix, treated with osmic acid, did not include any nervous tubes.

3. Primary, transverse myelitis reproduces, with some modifications, the tableau that I have presented to you of myelitis by compression, and here also the syndrome, *spasmodic paraplegia*, may be, according to the preceding conditions, connected with a consecutive descending sclerosis.

But *spasmodic paraplegia* may, quite frequently, be seen, clinically, under a form which hangs on for quite a long time and which has not been studied as much as it merits. Here the patient is not, as in the greater part of the cases heretofore supposed, condemned to stay in bed. He can often from the origin of the disease walk without support and even for lengthy distances. But the manner of walking is entirely peculiar. Olivier (d'Angers) has made a faithful picture of it in his description of chronic myelitis; and quite recently too, Erb has made a minute study of it and designates it as a spasmodic gait (*spasticher Gang*). Seguin qualifies the same syndrome as *tetanoid paraplegia.*

When the patient is lying down the stiffness of the members is already very sensible; when he is seated it is still more manifest; the legs are extended on the thighs and the feet suspended in the air, making it impossible for the patient to rest them on the ground. In fine, from the first movements for walking, "the trunk becomes erect and is thrown backwards, as if to counter-balance the weight of the leg, which trembles involuntarily until it can be rested again on the ground." The foot is seized with a trepidation each time that it is carried forward and the trembling for a moment extends to all the body.

It is doubtless this form of paraplegia that is related most frequently to common spinal lesions, myelites, compression, etc.; moreover, besides the rigidity of the members there exist still other concomitant or anterior symptoms, which admit of no doubt in that regard. But in other cases the disease dates from infancy, or rather is developed, slowly and progressively, in the absence of all symptoms other than muscular rigidity, which, from the inferior members where it remains, frequently, confined, tends at

times to gain the superior members. Erb has emitted the idea that these cases are related to a special pathological form, which he proposed to characterize as *spasmodic paraplegia.* He has considered it quite reasonable even, that such an affection is related to a primary sclerosis of the lateral columns. I have shared, and still do, the opinion of Erb in what concerns the popular character of a number of the cases which, clinically, affect that form of spasmodic paralysis; and I have proposed to unite them in a particular nosographic group, under the name of *spasmodic dorsal tabes.*[1] Thus, spasmodic dorsal tabes would be a particular disease; and spasmodic paraplegia would represent a syndrome common to many diseases—to spasmodic tabes amongst others.

But I am the first to assert that spasmodic dorsal tabes, regarded as a distinct nosographic species, should not have a definite and real recognition until pathological anatomy shall have spoken in favor of its autonomy. If it really concerns a distinct affection, the autopsy should reveal an equally special lesion—possibly a primitive sclerosis of the pyramidal fasciculi, suspected by Erb. If, on the contrary, the necroscopy demonstrates that, sometimes it is related to a myelitis by compression, and again to syphilitic myelitis, or something else, it is clear that the clinical autonomy is only in appearance.

The question then is not yet decided. I will only remark that the first results of the anatomico-pathological proof are not favorable to the doctrine of the morbid unity of spasmodic dorsal tabes. In fact, some cases that I have attached to this nosological group, have been, after the autopsy, detached and united to an affection for a long time known as sufficient to give rise to symptoms of spasmodic paraplegia. I refer to multilocular sclerosis (*sclerose en plaques*); but that is a point that should be studied with certain details, and which our time to-day does not permit us to undertake.

1 See on this subject the Thesis of M. Betons. "Etudes sur le tabes spasmodique."

LECTURE SIXTEEN.

TRANSVERSE MYELITIS.—SPASMODIC DORSAL TABES.

Summary:—Further Considerations of Organic Spinal Affections, with Contracture Localized.—Transverse Myelitis, and Hemilateral Section.—Intercrossing of Fibres of the Pyramidal Fasciculi.—Descending Sclerosis may Cross to the Opposite Side.—In Paralysis both Sides are Affected.—Total Transverse Myelitis.—Symptoms in incomplete Cures.—Spasmodic Gait.—Is Spasmodic Paraplegia a unique Disease?—Chronic Transverse Myelitis.—The Spasmodic Spinal Paralysis of Erb a Systematic and Symmetrical Lesion of the Lateral Fasciculi.—The Lesion of Spasmodic Dorsal Tabes of Charcot not yet Defined: Disseminated Sclerosis may be Mistaken for it.—Chief Diagnostic Symptoms in Spasmodic Dorsal Tabes: Exceptions.—Sketch of Morbid Conditions of the Disease as seen in the Adult and in Infancy.—The Entity of the Disease is Disputed by many Observers.—Thus far no Anatomical Substratum has been Demonstrated.—Its Diagnosis Difficult.

Gentlemen:

I propose to continue and terminate to-day the review of organic spinal affections in which permanent contracture of the paralysed members is an habitual symptom appertaining to the classical description of the disease. My object, as you are aware, is to make you recognize that the constant regular existence, or nearly so, of a lesion, either primary or consecutive, of the pyramidal fasciculi is a trait common to all the diseases in question. In the course of this exposition which, at first view, seems to involve principally a question of pure theory, we have encountered already, and shall still encounter, data of a certain practical bearing and of which you will very often find the application in the clinical study of cerebro-spinal diseases.

I. 1. Our attention is attracted very particularly to transverse myelitis, and we have considered cases in which the lesion occupies, at a given point, the entire mass of the spinal column. I must say a few words relative to those cases in which the trans-

verse lesion occupies one part alone of the spinal mass and in such a manner as to reproduce in some degree the lesion designated in experimental pathology as the *hemilateral section*. This order of spinal localization in focus is seen very often, as I have already remarked to you, in practice. It is not rare that the alterations of myelitis by compression, syphilitic, traumatic or spontaneous, may be the focal hemilateral lesion.

In this category of facts, I will remind you of two examples chosen from among many others for purposes of illustration: 1. The case of a traumatic lesion by a knife, cutting through, transversely, one-half of the spinal cord; 2. The very common case of a spontaneous syphilitic myelitis. Here the lesion involves at the same time one of the columns of the gray substance, the posterior fasciculi and the antero-lateral fasciculi of the same side, but the essential point that I desire to raise is, that the consecutive lesion of the lateral column is a descending lesion, and we know that the degenerative lesion is due to an interruption of the course of the fibres of the pyramidal fasciculus.

2. Already, gentlemen, I have had occasion to point out a fact that has been observed in many cases, namely, that the descending sclerosis is not always limited to the corresponding side,—that it extends, occasionally, to the opposite side; as in the case of Muller, of which I have given you the principal details.

To explain this fact, so singular in appearance, I have proposed the hypothesis that some of the fibres of each of the pyramidal fasciculi which have already intercrossed in the inferior part of the bulb, undergo in the cord itself a second decussation, at least so in certain subjects; and it is necessary to admit, in my hypothesis, that the twice intercrossed fibres are not interrupted in their course by a ganglionary cell; and, that coming from the pyramidal fasciculus of the right side they proceed to form a part of the pyramidal fasciculus of the left side.

This theory is founded, principally, at this time, on the anatomico-pathological fact in question; it will not therefore be uninteresting to see if it be sustained by some data of normal anatomy.

Quite a number of authors, among whom are Kölliker, Gerlach

and Krause, describe in the anterior commissure, an intercrossing in which fibres from diverse parts are concerned. But all agree to recognize, that fibres coming from one of the cornua of the gray substance traverse the median line and join the anterior fasciculus of the opposite side. In these descriptions it is not declared that the nervous fibres put in direct communication the pyramidal fasciculus of one side with that of the other. Nevertheless by the aid of preparations made with the chloride of gold, Schiffendecker, who has studied this matter with great care, assumes to have recognized nervous fibres, which, pointing from the right pyramidal fasciculus, run directly to the anterior commissure, and which, from the front of the central canal, may be followed to a certain distance on the other side of the median line.

Do these fibres penetrate the anterior fasciculi, or are they arrested in the gray substance? Or do they pass, on the contrary, into the opposite lateral columns? That is not demonstrated; however, it is not impossible. I doubt that normal anatomy, alone, can decide the question; but it is not unreasonable to suppose that in pathological states the tract of the degenerated fasciculi may be followed out; and if the supposed disposition really exists it will explain not only the fact, perfectly established, of a descending sclerosis of the two pyramidal fasciculi in the case of a unilateral lesion, but also the fact always recognized in experimental physiology, that a transverse hemilateral lesion produces motor paralysis as well in the opposite member as in the one corresponding to the section.

On this point, I will remind you also, that the experiments of Schiff and Vulpian have led to a modification, in this regard, of the traditional doctrines which go back to the time of Galen. It was believed that the transmission of voluntary incitations by the white fasciculi was exclusively direct. Now we know that though it is in the main direct, nevertheless it is in part crossed. In other terms, the section of one lateral moiety of the cord in a guinea pig, for example, produces a paralysis of both sides; but much more pronounced, certainly, on the side of the lesion than on the opposite side.

3. It is in this way, very nearly, that some of the fibres take their course in man, at least in a certain number of individuals. The paralysis of the side of the lesion is never as complete as might be supposed if the transmission of the voluntary incitations were only direct; and on the other hand, it is rare that the member of the side opposite to the lesion does not present to some extent also a condition of paralysis. This hypothesis offers, then, certain advantages, since it still allows locomotion, when even the hemilateral lesion is very profound, by distributing the paralysis in some sort to both sides. For the rest, in the ulterior course of events, the tendinous, cutaneous and other reflexions, the rigidity and contracture should appear, in such a case, as absolutely, as if it concerned a case of total transverse myelitis. Only it is quite rare that the phenomena, under such circumstances, are very accentuated; and, other things being equal, they are much more pronounced on the side corresponding to the lesion.

II. I resume, now, after this digression, total transverse myelitis. We paused, you remember, to study the diverse modes of termination that paralysis may present in a similar exigency, when already permanent contracture is established. I remarked to you, that along side of cases completely cured there are cases to note where the cure is imperfect; the movements reappear in the inferior members, thanks to the amendment of the muscular rigidity, but this rigidity persists nevertheless to a certain extent, and though the patient may leave his bed and walk it is only by slow and painful steps. Consider, now, the situation in which the supposed patient is placed. Thus, as I said to you in the preceding lecture, when he is in bed the rigidity though considerably diminished, still exists to a certain degree; moreover, the tendinous reflexions are much more pronounced than in the normal states, and spinal trepidation supervenes on the slightest voluntary movement. When he takes a seat on a high chair his legs have a tendency to assume a horizontal position, and the feet do not touch the floor. When he attempts to rise and stands erect, both legs stiffen, and cling together, and are seized at the same time with epileptoid trepidation. At first he seems glued to the floor and it requires an effort to lift his feet in the attempt to walk.

Here let me present to you the remarkably faithful description given by Ollivier (d'Angers):

"Each foot," he says, "is lifted with difficulty from the floor, and to complete this effort and push it forward the trunk is straightened and thrown backward as if to counterbalance the weight of the inferior member, which is seized with an involuntary trembling before it can rest again on the floor."

"In these attempts at walking, sometimes, the point of the foot is depressed and drags more or less on the floor before it can clear it, again it is raised brusquely and at the same time the foot is thrown outward. I have seen patients who could not walk a step, although resting on a cane, except by throwing backwards the trunk and the head in such a manner that their posture had some analogy with that which exists in tetanus."

All that, gentlemen, is exact, though it may be slightly over colored which is necessary in the description of a type. This type moreover presents a variation. The patient, in such a case, uses generally two crutches or canes, and walks, literally, on the points of his feet on account of the exaggerated contraction of the gastrocnemii. His body is thrown forward in an inclined plane in such a way that he is in danger every moment of falling headlong on his face. This variety, described by Erb, is more common than the type of Olivier (d'Angers).

It is very remarkable that this striking description of Olivier, and which is only applicable to exceptional cases, should have rested in a manner, as a dead letter until these later times. It had been overlooked at the epoch even when Duchenne described so carefully the character of locomotion in ataxia; and so completely so, that it seemed like revelation when Seguin (of New York), first, in 1873, then later Erb (of Heidelberg), in 1874, called anew our attention to the peculiar gait in certain forms of paraplegia, and which they propose, the one, to designate as *tetanoid paraplegia;* the other as the spastic gait (*spatischer Gang*).

You comprehend, gentlemen, that notwithstanding the interest attached to the act of spasmodic walking, we are not able to see in it the character of a particular disease; contrarily to what exists in the ataxia gait which is to a certain degree pathognomonic. It

is, in fact, a symptom common to several spinal diseases, and to arrive at a nosographic diagnosis, it is necessary to make account of the concomitant symptoms.

III. We have supposed thus far a case of transverse myelitis, acute or subacute in the beginning, where the symptoms attain rapidly the highest degree, and, then retrograde. But it must be kept in mind that there are cases in which the disease assumes from its origin the appearance of a primary chronic malady. The lesion in this case is still transverse, but it is incomplete and its evolution is slow. The patient, for example, has not been confined to the bed; it is a paraplegia in which the paretic symptoms have for a time, more or less long, been unperceived. But the gait has the spasmodic character from the onset, and however slow may have been the evolution of the disease, we must wait, as a rule, to see the paraplegia associated with other necessary symptoms to characterize, nosographically, the nature of the affection.

IV. Nevertheless, gentlemen, there exist, clinically, and it is not rare to encounter them, certain cases where the symptoms of spasmodic paralysis, developed primitively as in the preceding case, is seen in some sort isolated from every other symptom from the beginning to the end of the disease, without alteration of sensibility, or functional troubles of the rectum or bladder, or pseudo-neuralgic pains or cephalic concomitants. The affection is also particularly characterized by a slow evolution and by a marked tendency to invade, progressively, the superior members.

This order of spasmodic paraplegia has appeared special enough to some physicians—and I am of the number—to have led them to believe that it was not a case of ordinary transverse myelitis (by compression, syphilitic or otherwise), accidentally devoid of their ordinary attributes and revealed, as has been said, in an obscure form, but that it concerns a particular affection—an autonomic malady—and related very probably to a special localization.

Erb was among the first to adopt this belief in 1875. I followed him very soon, as my lectures in 1876 bear witness.

Erb has designated this assumed special affection as *spasmodic spinal paralysis.* I have proposed, since it concerns a particular morbid state, the name of *spasmodic dorsal tabes;* the term spasmodic paralysis representing only a syndrome common to many spinal diseases. The description given by Erb does not differ moreover, in any essential trait, from that which I gave subsequently to it. I do not differ from Erb except on one point. He has affirmed, or nearly so, that the lesion to which the symptoms belong is actually known—that it is a systematic and symmetric sclerosis of the lateral fasciculi. While recognizing the localization proposed by Erb as quite reasonable, I reserve my full opinion. I have remarked that all the observations of symmetrical sclerosis without participation of the anterior gray horns are quite old. "They are," I said, "old faded souvenirs that he would brighten once more. We must await the result of new observations before assuming opinions on this subject."

Up to this time, gentlemen, as I have just shown, anatomico-pathological anatomy has not furnished any definite proof, and so the solution of the problem remains in suspense. But while thus indeterminate, the clinical descriptions must be taken for what they are worth.

V. But before resting in such a conclusion I wish, conforming myself to what I announced in my last lecture, to say something to you about a cerebro-spinal disease quite well defined now, anatomically and clinically, and which, in some of its defaced forms, may appear, almost exclusively, as a spasmodic paraplegia of such a character that under that appearance the affection in question may be and has been, in fact, confounded with that which I have called *spasmodic dorsal tabes.* I refer to *disseminated multilocular spinal sclerosis.*

I will merely remind you that sclerosed foci, disposed without any order in various parts of the cerebro-spinal axis, predominate generally in the cord, where they occupy, by preference, the antero-lateral fasciculi. It is important now to state a fact to which I have already referred. It is that sclerosed patches when once developed will remain isolated in the fasciculi of the cord without giving rise to a secondary degeneration; this is a flagrant excep-

tion to the Wallerean law. It is of little importance, however: let it suffice for the moment to consider the affection exclusively on its clinical side.

First, we are obliged to show, as should be expected, from the multiplicity of lesions and the variability of their seat, that the symptoms of this disease are likewise very numerous and varied: cephalic troubles, such as nystagmus, amaurosis, embarassment of speech, vertigo and intellectual disturbances; spinal troubles, among which it is proper to mention as most common, a particular trembling of the superior members, sometimes replaced by contracture, spasmodic paraplegia, etc. I pass also muscular atrophy, which is also seen in a case where the gray substance is attacked; and the tabetic symptoms when there exist lesions of the posterior fasciculi. Such in short are the signs which permit us when they are found united, to readily diagnose the disease.

But it may happen that the combination is decomposed piece by piece in such a manner that many of the symptoms will be absent. Thus it may be that only the cephalic troubles, vertigo, nystagmus, etc., are observed. Again, on the contrary, there will only exist symptoms of spasmodic paraplegia nearly isolated; I say *nearly isolated*, for after a careful scrutiny one discovers the actual or past existence of some one of the concomitant phenomena which are seen so largely in typical cases, also, in presence of a spasmodic paraplegia, it is necessary to bear in mind the series of symptoms which may be found united in a case of complete multilocular sclerosis. It must be quite exceptional if two or three among them do not appear associated at a certain stage of the disease, with spasmodic paralysis, if this depends on a disseminated sclerosis, and it is especially in this regard, that the diagnosis may be established.

VI. Now, gentlemen, I proceed to present concisely a sketch of the morbid state that both Erb and myself believed it proper, until possessed of more ample information, to consider as a particular disease—*spasmodic dorsal tabes:*

1. The clinical description may be traced moreover in a few words. The disease is developed in persons aged from thirty to

fifty years, particularly among men, and in the absence of all appreciable occasional cause. There is no disturbance of sensibility; it developes slowly and progressively; manifesting itself at first by a simple heaviness of the legs; then by a veritable paresis accompanied with stiffness; at length the gait takes on the spasmodic character and the patient is often obliged to keep his bed; but sometimes only after many years. As it takes its course the tendinous reflexions become very exaggerated, while the cutaneous reflexions present their normal character.

2. The disease as seen in infantile life merits special attention (Erb, Seeligmüller, Stromeyer). Sometimes the rigidity begins to appear soon after birth without accompaniment of cerebral symptoms. The nurse perceives that the members are rigid and that henceforth it becomes more difficult to dress the child. Sometimes the trunk itself is rigid. Coming to the period of walking it is then observed that the erect station and walking are impossible. It is necessary to wait until the child is three or four years old to see it painfully attempt to stand erect, supporting itself by clinging to the furniture. The manner in which children of that age, sustained under the arms, progress, whether good or bad, is very characteristic. The hips are slightly flexed and the knees are drawn together with such force that the legs and feet clog each other in intercrossing. Finally, the plantar flexion of both feet, which rest on their toes, causes an inclination of the body forward, further obstructs locomotion.

Moreover, the tendinous reflexions are exalted. No muscular atrophy occurs; the muscles preserve their normal excitability; there exists therefore a well marked contrast, in all respects, between this affection and infantile spinal paralysis; and so we see plainly that parallel with infantile spinal paralysis there is an infantile spasmodic paralysis, and very distinctly and clearly separated from it.

In their turn the superior extremities become affected; the forearm stiffens, is half flexed and in pronation; the fingers are folded upon the palms. But never, I repeat it, do we observe cephalic complications, and the vertebral column presents nothing

abnormal. The pathogeny of the disease is consequently extremely vague. Seeligmüller proposes, premature labor at seven or eight months, and consanguinity. But these are pretexts rather than reasons. Finally, autopsies fail. One cannot nevertheless suppress the idea that at the period when the disease begins, the lateral fasciculus being in full course of development, the condition, under certain influences, is not unfavorable to the production of an inflammatory lesion.

3. In adults a lesion of the same order, equally limited to the lateral fasciculi, would readily account for the whole of the phenomena. But, again I say, up to this time the hypothesis has not received verification. Here then is an interesting problem in pathological anatomy to resolve, and I cannot too earnestly engage you to give it your attention, if it shall happen to you to have a case of the kind in your charge.

4. I have just said that spasmodic tabes has as yet only a clinical existence, but if in reality, as I believe, it concerns a morbid species, it thus far entirely fails of an anatomical substratum. Nevertheless, latterly, a certain number of authors have attempted to demonstrate that this disease is only an artificial nosographic construction, and that a lesion of some form or other of myelitis, spontaneous, compressive, or syphilitic, can give rise to this ensemble, which should no longer represent therefore a special affection.

In support of this theory there have been published observations which have led some to believe that in cases where they have recognized the characters assigned by Erb and myself to what I call spasmodic dorsal tabes, they have seen at the autopsy the most variable spinal lesions. I have examined these observations with great care and I cannot perceive that any of them really possess the signification attached to them.

On the clinical side there are cases of common myelitis by compression, syphilitic in fact, abnormal in some respects, but where we always recognize more or less accentuated, the troubles of sensibility, of the functions of the bladder and rectum, so characteristic in this form of myelitis; at the autopsies would be seen diverse lesions and presenting always, this common

trait essential to the species, that the alterations in question are associated with a lateral sclerosis. That only proves what is well known—that the spasmodic gait, or, if you like better, the spasmodic paralysis, may exhibit itself under the most varied forms of myelitis. But in the morbid species the gait is not all; it is only an element of the disease.

That demonstrates also that the diagnosis is difficult and that before deciding it is necessary to scrutinize closely. I have found myself mistaken at least once, I frankly acknowledge it, in a case presented at my clinic as an example of spasmodic tabes. The autopsy demonstrated that it was a case of disseminated sclerosis. But on reviewing the observations we perceived that the patient had had vertigo, with tremblings of the extremities; valuable symptoms which should have indicated the way to a true diagnosis. Since that time I have equally traced to their legitimate origin during life conditions which I regarded as indicating spasmodic tabes; and the autopsy confirmed my diagnosis.

Thus matters remain as they were before the publication of the adverse observations to which I have just made allusion; and now, to sum up, if in default of sufficient anatomical observations the nosographic, autonomous existence of spasmodic dorsal tabes is not yet solidly established, it may be said, on the other hand, that notwithstanding what the critics have said, it is not yet seriously shaken.

You see then, gentlemen, by the preceding exposition that permanent contracture is a symptom common to spinal organic affections (and they are numerous), where there exists a lesion of the lateral fasciculi. It is, however, important to remember that permanent contracture is not a certain indication of an organic lesion of the spinal marrow, for there exist many cases where the lateral fasciculi are possibly affected functionally, but, indubitably, without a material lesion. Under this head I will cite hysteria, which, as I have often said, is one of the most demonstrative examples.

LECTURE SEVENTEEN.

SPINAL AMYTROPHIES.—LOCALIZATIONS IN THE GRAY SUBSTANCE OF THE SPINAL CORD.

Summary:—Systematic Lesions.—Gray Substance of the Cord.—Its distinct Regions.—Center of Reflex Actions and Route of Transmission of Sensory and Motor Influences.—Systematic Anterior Poliomyelitis.—First Class: Acute Protopathic Spinal Amyotrophies.—Infantile Spinal Paralysis.—Adult Spinal Paralysis.—Subacute Anterior Poliomyclitis.—Chronic Systematic Anterior Poliomyelitis, or Protopathic Progressive Spinal Amyotrophy.—Second Class: Acute Diffused Poliomyelitis.—Chronic Peri-ependymar Sclerosis.—Hypertrophic Spinal Meningitis.—Multiple Sclerosis.—Amyotrophic Lateral Sclerosis.—Solidarity of the Anterior Motor Cell, the Centrifugal Nerve and the Muscular Fibre.—Discoveries of Doyere and Rouget on the Termination of Motor Nerves in the Muscular Substance.—The Nervo-Muscular System.

GENTLEMEN:

You certainly have not lost sight of the tableau, or better, the topographic plan, that I presented to you at the inauguration of my course for this year, and which I have had occasion so very often to lay before you. It was intended as you well remember, to show at a glance the diverse regions of the spinal cord in which, by a sort of selection, are cantoned the spinal lesions that are now called *systematic lesions*.

That denomination, *systematic lesions*, which I borrowed from the teachings of Vulpian is, I have remarked to you many times, perfectly appropriate. In fact, these departments—these regions that disease may invade in detail, without participation of the neighboring regions, represent so many *systems*, anatomically and functionally distinct; they are, in a manner, so many organs, each one of which plays its particular physiological part, and consequently, according to the logic of facts, the affection of each one of these organs in pathological conditions should be construed by

a proper symptomatology. Therefore the symptoms being known, it will be possible for the clinician to ascend to the lesion and determine its seat.

We are, very naturally, led by the preceding considerations to regard systematic lesions in the domain of the spinal cord, as so many elementary affections, the profound knowledge of which should be applied to the clearing up of more complex affections, non-systematic, or in other terms, distributed in the nervous column in an unequal or diffused fashion; that is to say, to be methodical, the study of systematic spinal lesions should necessarily precede that of spinal lesions non-systematic.

I dare hope, gentlemen, that the developments into which we have entered apposite to systematic lesions of the pyramidal fasciculi have justified, in part, these propositions which I have so frequently laid before you. They have enabled you likewise, more or less, if I am not mistaken, to understand the true signification of the syndrome designated as *spasmodic paralysis*, which as you have seen, plays so predominant a part in spinal pathology.

I. To-day I desire to direct your whole attention to a region that we have already entered upon several times, incidentally, in the progress of our studies, but which has not been as yet an object of regular exploration. I allude to the spinal gray axis, or in fewer words, the gray substance.

It is unnecessary to show you again, that though it occupies in the cord a space relatively small, this gray substance is, nevertheless, in a physiological point of view, the most important part of the spinal centre. It will suffice to recall that it is the essential place, in fact, for the transmission of the sensitive impressions, and motor impulsions, voluntary or reflex; so that if this path way be severed, the accomplishment of all these functions would at once become impossible.

Moreover, it seems now peremptorily demonstrated that all parts of the gray substance are not indistinctly affected in the execution of these diverse functions. In this space so limited, it is possible, in fact, to establish physiologically, several quite distinct regions. Thus, in this regard the central gray substance should be distinguished from the horns or columns of the gray substance. The

first alone, with the posterior horns to a certain extent, has for function, the transmission of sensitive impressions; while the anterior horns seemed destined exclusively, for the transmission of motor impulses, and have no relation with sensibility.

II. These results, obtained by experiment have been confirmed in the domain of pathology. Disease, indeed, better than the most skilfull physiologist, produces alterations which affect, isolately, certain regions of the gray substance.

A. Now, gentlemen, we have here a capital fact in the history of the systematic lesions of the gray substance. It is that those alone, that merit truly the name that all recognize, are found located in the same region always, and that region is the anterior horns.

The diseases in question have then, you observe, as a univocal, anatomical character, not only to be circumscribed in the anterior columns, but even more; to constitute an exclusive lesion, systematic in the rigorous acceptation of that word; that is to say, confined in that region, without participation, except accidentally, with the adjoining regions.

The lesions which appertain to this narrow localization, are nearly always of an irritative, or inflammatory nature. Latterly it has been proposed to distinguish them by a term which, at once, indicates the nature of the morbid process, and the nature of the localization; hence the term *systematic anterior poliomyelitis;* systematic, because the anterior gray substance may be affected incidentally, or secondarily, in the case of spinal affections of another order. It is necessary also to add the qualification *acute* or *chronic*, accordingly as the affection is evolved in the one, or the other of these two modes.

By reason even of this localization, and also on account of the physiological specificity of the regions interested, the affections of this group, as may be readily anticipated, are seen clinically with a certain number of common and distinct traits which distinguish them from all the spinal affections which occupy, in the medullary columns, a different seat.

B. In a few words, here are the fundamental characters of the group:

1. The muscles are stricken with motor impotency. The paralysis is more or less complete; but—and this is a fundamental fact—the affected muscles are, besides, the seat of trophic lesions more or less profound and particularly made manifest by special electrical reactions. This is a fact that distinguishes, at a glance, these paralyses from those which result from a lesion of the white fasciculi, in which case the nutrition of the muscles is not at all affected.

2. The muscles of animal life are alone interested or, at most, the muscles of the bladder and the rectum may remain unaffected. The electrical qualification just referred to is sufficiently distinctive, for you to carefully note. It is not easy at this time to give a satisfactory explanation of it.

3. There exist, in these affections, no modification, except incidentally, of sensibility. This feature distinguishes systematic affections from those which occupy in a diffused fashion the gray substance; and these last, besides troubles of sensibility affect a marked tendency to cutaneous trophic troubles, eschars, etc., a tendency which is never seen in systematic lesions.

4. In most cases, more especially where it concerns the acute and subacute forms, the various reflexions are either diminished or abolished completely. You have foreseen that spinal epilepsy, no more than contracture, does not appertain to affections of this group; only by reason of the unequal distribution, often, of the trophic muscular lesions, you will encounter under such circumstances, paralytic deformations or deviations.

III. A. The systematic, anterior poliomyelites form, in the main, a nosographic group quite natural. The dominant, unique, exclusive symptom is, as you are aware, the trophic muscular lesion. Also these alterations have been sometimes designated as *spinal amyotrophies* or of *spinal cause*. But it is proper to add the qualitative *protopathic*, which indicates that the lesion of the spinal region, whence is derived the muscular lesion, is the fundamental fact. On the other hand some will call *deutero-pathic spinal amyotrophies*, the various affections of the spinal cord in which the lesion of the anterior horns is only secondary or accidental, and where the trophic alteration of the muscles is

found, consequently, intermingled, clinically, with the other symptoms.

B. The group of systematic anterior poliomyelites includes affections which appertain to the daily clinic, and to which the physician should give especial interest. I believe it proper, therefore, to refresh your minds by a concise enumeration of them.

As I have said the systematic anterior spinal lesions are evolved sometimes in an acute, and sometimes in a chronic manner. I shall include in this enumeration only the species in regard to which, the pathological anatomy is at this time definitely ascertained.

I. A first class is composed of the group of *acute protopathic spinal amyotrophies.*

(*a*) The species *infantile spinal paralysis* is a disease that has been especially studied by Duchenne of Boulogne and Heine. It is, of the group now in question, a model disease for anatomico-physiological study. Indeed the lesions are perfectly circumscribed; again the symptomatology is itself very limited and precise, and nearly all the details that it admits of, may be to-day easily interpreted in the light of pathological facts.

(*b*) *Spinal paralysis of the adult* is in some degree the same disease transported into the pathology of the adult. For a long time the relationship had been established, uniquely, on the faith of a symptomatology veritably very special; but now the anatomical control is definitely pronounced in favor of the connection. It is not so, up to this time, of the second group of which I shall limit myself to a mere mention.

2. *Subacute anterior poliomyelites.* These respond to the affection described by Duchenne, under the name *subacute anterior spinal general paralysis.* This is still, on more than one point, a chapter of expectation, for, I repeat, the anatomico-pathological observations relative to this form have not as yet furnished peremptory results.

3. Finally, *chronic systematic anterior poliomyelitis* is represented by the form of amyotrophy, of which Aran and Duchenne have given the clinical description, and to which they have given the name of *progressive muscular atrophy.* Cruveilhier recognized in this affection a lesion of the spinal anterior roots. Modern researches have demonstrated that this alteration, mentioned and

described by Cruveilhier, is connected with an irritative systematic lesion of the anterior gray horns. The disease in question has been also designated sometimes under the name of *protopathic progressive spinal amyotrophy*.

IV. Such, gentlemen, are the great species of the group of the amyotrophies related to anterior poliomyelitis. In juxtaposition, were it only to establish a comparative term and form a contrast, it is proper to exhibit for a moment various spinal affections, in which the lesion of the anterior horns may, doubtless, exist, but not constitute the capital, unique anatomico-pathological fact.

In such a case the original lesion is without the gray substance, at least so in regard to the region of the anterior horns, which are found affected only consecutively, by extension. But the trophic alteration is, nevertheless, developed by reason of the participation of the anterior horns. Then it is seen that this symptom is, in a manner, superadded to those of the principal disease, and in such a case the clinician should expect to see developed a complex symptomatic whole; for, truly, there is, perhaps, not an acute or chronic spinal lesion which may not, at a given moment, invade the anterior horns and determine there the lesion of the ganglionary elements, whence is derived spinal amyotrophy.

Not to pause longer at these general and necessarily slight and vague remarks allow me to invite your attention to a certain number of concrete cases.

1. (*a*) Among the non systematic diffused spinal lesions may be cited in the acute form, central myelites or diffused poliomyelites. Here the trophic lesion of the muscles, analagous to infantile paralysis, is a frequent occurrence. But there exists concomitantly a disturbance of sensibility, a derangement more or less profound of the functions of the bladder and of the rectum, a development of eschars, etc., and if the patient survive, and the white fasciculi participate in the anatomico-morbid process, there is developed a permanent contracture, united to all the other signs of spasmodic paralysis.

(*b*) In the chronic form I will cite *peri-ependymar sclerosis*; *hypertrophic spinal meningitis*; and lastly *multilocular sclerosis* (*sclerose en plaques*), which, under certain circumstances, may assume the

appearance of a progressive amyotrophy. There exist even non-inflammatory lesions which may have an identical result, such as hydromyelus and intra-spinal tumors (glioma, sarcoma, etc.)

2. Among the systematic lesions I must mention posterior sclerosis which, in quite a number of instances, extends into the gray substance. But the morbid form to which, in this enumeration, I wish especially to call your attention is *amyotrophic lateral sclerosis*. This affection, as I have told you, includes two anatomico-pathological elements, viz., a lesion of the lateral fasciculi and a lesion of the anterior cornua. And this is not accidental; it forms, in some sort, an integral part of the disease, though, really, it is developed, all leads us to believe, secondarily. It concerns then a systematic lesion of *combined elements* as the Germans say. It is that form which I have thought proper to study in detail with you on several occasions already, because it shows that the laws of localization formulated apposite of the gray spinal substance are reproduced in the bulb. You know, indeed, that the nuclei of origin of the hypoglossal and the facial nerves, which represent the anterior cornua in the medulla oblongata, are often the exclusive seat of pathological lesions, of such sort, that there exist protopathic bulbar amyotrophies, which may be contrasted with deuteropathic bulbar amyotrophies.

V. This rapid view of the whole field will enable you, gentlemen, to examine much closer the subject, by placing you in the point of view of anatomy and of pathological physiology. It will become necessary, in other terms, when you study this subject to direct your attention, particularly, to the gray substance whence are derived the symptoms of amyotrophy. But here, as everywhere else, the anatomico-pathological investigation in order to be fruitful, presupposes, necessarily, a profound knowledge of the normal conditions; and, without being willing to enter, understand, into all the details which relate to such a subject, I will point out to you, especially, some things belonging to the anatomy and the physiology of the regions you will have to explore. Not only in this preparatory study must you consider the gray substance itself, but also the motor nerves which originate there, and the striated muscles to which they transmit motor force.

Indeed, gentlemen, the various parts just mentioned are, in certain respects, a solidarity; anatomically and physiologically they represent a system. The motor nervous cell with its multiple prolongations may be truly considered as a small independent organ; it forms the bond which unites several systems to one another; but does not appertain exclusively to any one of them.

Nevertheless, it is important to remark that of all the prolongations of the anterior spinal cell, the most important, the most characteristic, in the double point of view of physiology and morphology, is the one which puts the cellular organ in direct continuity with the motor nerves, and in such a manner, that the motor nervous tube in its axis cylinder, which is its essential part, is only an emanation of the substance itself of the motor cell. To-day, we know, in fact, that by its peripheral extremity, this prolongation of ganglionary element enters into immediate relation, so to speak, with the muscular element. Previous to 1840 on the faith of the researches of Valentine and Burdach, it was thought that the peripheric extremities of the muscular nerves terminate in loops in the intervals of the primary muscular fasciculi. But Doyère made a capital discovery, when he demonstrated that among the tardigrades, the motor nerve terminates by a unique filament on the level of an enlargement which forms a part of the primary fasciculus, and which is yet called, the eminence or hillock of Doyère.

You all know, too, that in 1862 Rouget went further, and proved that under the sarcolemma, that is, in the substance even of the primary fasciculus, the eminence of Doyère is formed by a mass of granular substance in which terminate the nervous tubes, reduced to their axis cylinder. The labors of Krause, of Kühne, and also Ranvier, have confirmed these data in a general way, by the addition of a mass of important details. But the grand fact discovered by Rouget is, justly, that close connection of the centrifugal nerve with the muscular substance.

Thus you see, there exists, on one hand, between the ganglionary element and the axis cylinder, which is only a prolongation of that element, a relation of immediate contiguity; the same

exists on the other hand, between the terminal extremity of this prolongation and the substance of the muscular fasciculus.

There is, then, as I have announced, anatomically speaking, an intimate solidarity between the motor cell, the motor nerve, and the muscular fibre; really, we can consider them as three connective elements of the same system; and, even, say without straining a point, that the spinal cell plunges directly into the substance of the muscular cell by the intermediation of its axillary prolongation.

But it is important to remark, gentlemen, in this association, that one of the elements is, so to speak, dominant; the others are subordinate. The integrity of the muscles as well as the nerves depends on that of the ganglionary elements. It is demonstrated, indeed, that a lesion of the ganglionary element reacts necessarily on the muscle through the nerves; that the lesion of the nerve reacts on the muscle which, consequently, occupies the lowest rank in the association; but it is in no wise demonstrated, on the other hand, thus far at least, that a lesion of the muscles or of the motor nerves, can react on the ganglionary elements so as to disturb their nutrition.

Such are the considerations that I have felt impelled to present to you, gentlemen, on this totality of elements, which may be designated as the *neuro-muscular system*, and of which the preliminary study is indispensable to whoever wishes to successfully grasp the pathological topography of the various regions of the gray substance.

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